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THE ANATOMY AND PATHOLOGY OF PREMATURE SEPARATION OF THE PLACENTA*

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The voluminous literature on premature separation of the placenta indicates that, although much detailed histological work has been carried out on the separated placenta, there is little unanimity of opinion regarding the pathogenesis of accidental haemorrhage, placental infarction, and premature separation of the placenta in general. This is mainly due to the void which still exists in our knowledge of the anatomy and physiology of the placental circulation.

In this paper an attempt is made to review briefly some of the latest concepts regarding (1) the anatomy of the placental circulation, both foetal and maternal, with reference to its embryological development, and (2) some aspects of the pathology of premature separation of the placenta, particularly the lesions on the maternal side of the placental circulation.

ANATOMY OF PLACENTAL CIRCULATION

The Foetal Placental Circulation

Hertig and Rock have shown that at about 5½ or 6 days after ovulation the fertilized ovum is implanted upon the prepared endometrium.[1] On the 9th day the ovum is entirely imbedded in the decidua except for the operculum deciduae.[2] The syncytiotrophoblast is seen extensively invading the surrounding decidua and forming lakes or lacunae in the substance of the trophoblast.[1] These lacunar spaces at first contain leucocytes.[3] The trophoblast, by its proteolytic and cytolytic action, erodes the spiral arterioles and venules of the endometrium and the first decidual-trophoblastic circulation is established by the gradual replacement of the leucocytes in the lacunar spaces by a slow-flowing maternal blood stream. These spaces are therefore lined by a layer of syncytiotrophoblast which, in its early trabeculated form, is called the primary villus system on account of its resemblance to the future or permanent villi.

At about the 13th or 15th day after ovulation, bud-like outgrowths of cytotrophoblast appear to invade the lacunar spaces, carrying with them an outer layer of syncytiotrophoblast covering and an inner core of extra-embryonic mesoderm. These are the secondary or permanent villi, which by the 16th day form branching villi, containing vascular primordia from which are formed capillary blood vessels that will ramify over the internal surface of the trophoblast to link up with the foetal circulation *via* the vitelline vessels and establish the foetal placental circulation at about the 21st or 23rd day. Until these vessels unite, the chorionic tissues are nourished entirely by the maternal blood stream —a point in favour of the theory that the placenta is entirely dependent upon the maternal circulation for its nourishment.

As the ovum grows it extends into the uterine cavity, with resultant atrophy of the decidua capsularis and chorion laeve, until at the 12th week these two layers loosely blend with the decidua vera to obliterate the uterine cavity, while the future placenta is formed by the chorion frondosum, intimately attached to the decidua basalis.[4]

The villi can now be seen to consist of a double layer of trophoblast covering a loose stroma containing capillary blood-vessels. Compared with a villus at term it is seen that the nuclei of the syncytiotrophoblast have become clumped together as syncytial knots. The Langhans layer has disappeared and the stroma has become more fibrous and the capillary vessels larger, resembling capillaries seen elsewhere in the body.

All these changes are so-called degenerative changes associated with normal aging of the placenta.[4]

The placenta at term consists of a layer of amnion and a double layer of chorion and the blood vessels with their related cotyledons. Between the two layers of chorion run branches of the umbilical vessels—placental arteries and veins—which divide and subdivide either on the foetal surface or in the substance of the placenta, each to terminate in a cotyledon, of which there are approximately 200 in number, according to Crawford.[5] By further dichotomy these vessels divide to form sub-cotyledons, and finally by their finest subdivision form capillaries, which are imbedded in a stroma and covered by trophoblast—in other words, the villi.

By using a retrograde injection and digestion method to study the placental vessels histologically, Crawford[5] has shown a complex villus to be a three-dimensional structure with finger-like projections. He also noted the absence of anastomoses between villi.

The Maternal Placental Circulation

The maternal aspect of the placental circulation was described by Spanner[6] in 1936. He showed, by injection and corrosion methods, that the placental bed consists of a network of arterial and venous sinusoids underlying the placenta, and demonstrated clearly how the spiral arterioles enter the decidual-placental space. They enter the space from the whole of the under surface of the placental site. He could not, however, with rare exceptions demonstrate any venous drainage from the central area of the placental site. According to his findings, the venous return was by way of the

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marginal sinus. Spanner showed clearly the existence of this sinus as the peripheral part of the intervillous space, bounded laterally by the chorio-decidual reflection, superiorly by the chorionic plate and inferiorly by the peripheral part of the decidua basalis. He furthermore demonstrated the existence of venous sphincters in the foetal placental vessels both on the surface and in the substance of the placenta.

Ramsey,[7] in 1954, demonstrated that in monkeys the venous drainage of the intervillous space does take place through the central part of the placental site as well as through the peripheral or marginal-sinus area. This was demonstrated by injecting Indian ink into the uterine veins. The problem then arose how adequately to explain why blood should enter the intervillous space through the spiral arterioles and bathe the cotyledons and should not be returned immediately *via* the adjacent venous exits. Many theories were formulated. The solution came from the Carnegie Institute in Washington,[8] where morphological studies have led to the conclusion that the circulation in the placenta of primates is effected by a *vis a tergo* of the maternal blood pressure. This conclusion is based on the proposition that a sharp fall in blood pressure takes place between the uterine arteries and the intervillous space, and an additional fall in pressure between the intervillous space and the uterine veins. It is further assumed that the Braxton Hicks contractions during pregnancy further enhance this pressure differential by intermittently compressing the uterine veins, thus producing a temporary rise of pressure in the intervillous space, with abruptly increased drainage from the intervillous space following relaxation of the myometrium. This hypothesis contradicts the traditional belief that the myometrial contractions 'squeeze' the placenta like a sponge, expressing its content of blood.

The studies of intra-uterine pressure carried out by Alvarez and Caldeyro[9] on human patients at Caesarean section confirm this hypothesis and supply actual values for some of the components of the system.

Premature Separation of the Placenta

In considering the mechanism involved in premature separation of the placenta it will be necessary to recall that the placenta normally separates through the spongy layer of the decidua. After the expulsion of the foetus the uterine contractions tear the placenta from the uterus through the large vascular spaces of the spongiosa.[10] The contracted uterus effectively prevents bleeding from this vascular layer by tamponading the arteries supplying the area. When the placenta separates prematurely this tamponading effect cannot take place and bleeding usually occurs.

Placenta praevia. In the partial types of placenta praevia the bleeding is always at the marginal area and would involve mainly the venous return from the marginal sinus, with a better prognosis for the foetus than is presented by separation of the central part of the placenta, such as occurs in concealed accidental haemorrhage involving mainly the arterial supply to the placenta. The bleeding of placenta praevia is in fact just as much due to a premature separation of the placenta as is that of abruptio placentae.

Rupture of the marginal sinus. Haemorrhage from rupture of the marginal sinus arises when the inferior or decidual border of the sinus ruptures and, according to the extent of medial spread, may involve a considerable portion of decidua in this detachment. Fortunately the haemorrhage is usually of a lesser degree, and thrombosis occurs which produces a laminated clot in a distended area in the marginal sinus. The diagnosis of this condition depends on the finding of a macroscopic and microscopic thrombus in this sinus, which is continuous with the clot that usually adheres to the periphery of the placenta at the site of the rupture. These criteria must be satisfied in order to differentiate the condition from the rupture of the marginal area, which of necessity follows the normal separation of the placenta.

Toxaemic States

As premature separation of the placenta occurs so frequently in association with toxaemia of pregnancy, it will be necessary to correlate the pathological findings in these two conditions. Ever since 1914, when Young[11] drew attention to the occurrence of red infarcts in the placentae of eclamptic women, much interest has been taken in the vascular lesions of this organ. During the last 25 years Bartholomew[12] and his co-workers have repeatedly emphasized the importance of these infarcts as the characteristic lesions in eclampsia and pre-eclamptic toxaemia. These workers originally attributed their formation to obstruction of the placental vessels owing to excessive and injurious foetal movement. Later, in 1936, Bartholomew and Kracke[13] suggested that the hypercholesteraemia of pregnancy, associated with a subclinical maternal hypothyroid state, might account for the degenerative vascular placental changes that result in infarction, of which they recognize 8 macroscopic varieties, 5 of which are described as so-called toxic infarcts. Their latest theory is that spasm of the muscular sphincters of the placental vessels, as originally described by Spanner, leads to the placental infarcts, which they hold are responsible for toxaemia of pregnancy.[12]

Bartholomew emphasizes that failure to appreciate the consistency with which acute infarcts may be observed in the placentae of patients affected by acute toxaemia is due mainly to the examination of the placenta in the fresh state. He points out that specimens should be placed in 10% formal saline immediately and allowed to become fixed for 1–2 months.[12] He further believes that the extent and location, as well as the degree, of obstruction of the placental vessels, and the rapidity of autolysis, will determine whether the condition will be one of mild, moderate or severe pre-eclampsia or whether accidental haemorrhage will occur. It is, however, as well to remember that the so-called placental infarcts occur in 60% of placentae in normal cases. Other workers, such as Nesbitt,[14] failed to demonstrate an exact correlation between the pathological lesions and the clinical manifestations of toxaemia. Nesbitt simplifies the understanding of placental infarction by pointing out that the whole question depends upon the maternal nutrition to the villi and the aetiological factor of syncytial degeneration, the latter primarily consisting of premature aging of the placenta. A large laminated clot may form beneath the decidual plate and thus become not only an area of intervillous thrombosis but also one of premature separation. Such an area of placental separation may interrupt the maternal circulation, either arterial or venous, and lead to infarction or ischaemic necrosis of the placenta.

Javert and Reiss,[15] in 1952, attempted to differentiate between red infarcts and decidual haemorrhages, explaining the intervillous thrombosis on the basis of an escape of foetal blood through the damaged endothelial lining, and the

Fig. 1. See text.

absence of such thrombosis in premature separation of the placenta.

It is interesting to note that as far back as 1915 Williams[16] described degenerative lesions in the intima of the uterine arterioles in association with toxaemia. In 1950 Zeek and Assali[17] reported vascular changes in the decidua associated with eclamptogenic toxaemia, believing that these obstructive changes were due to acute atherosis of the spiral arterioles and venous lakes.

In 1953 Hertig[18] stated that the pathological changes in accidental haemorrhage were decidual necrosis or degeneration, explained on a vascular basis in toxaemia and on a fortuitous basis when hypertension was not involved. Hertig demonstrated morphological evidence of acute atheromatous changes in the decidual arterioles, with macrophages and fibrinoid degeneration of the intima, resulting in encroachment on the lumen. These lesions were also demonstrable in essential hypertension and chronic nephritis.

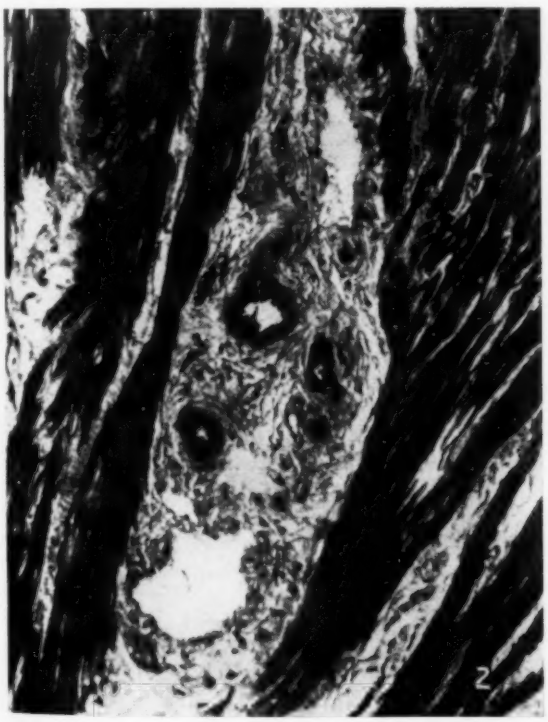

Fig. 2. See text.

Owing to the manifold difficulties in obtaining suitable material for study, Dixon and Robertson,[19] of Jamaica, have developed an ingenious method for obtaining material suitable for histological investigation of the vessels of the placental bed. To obtain representative specimens of decidual and underlying myometrial vessels they use a cervical punch-biopsy forceps, thrust through the placenta before separation at Caesarean section. With this technique they found vascular changes in the decidual and myometrial vessels from hypertensive and toxaemic patients similar to those generally acknowledged to occur in systemic hypertension. These changes were directly related to the clinical severity of the disease. Few such changes could be demonstrated in normal patients.

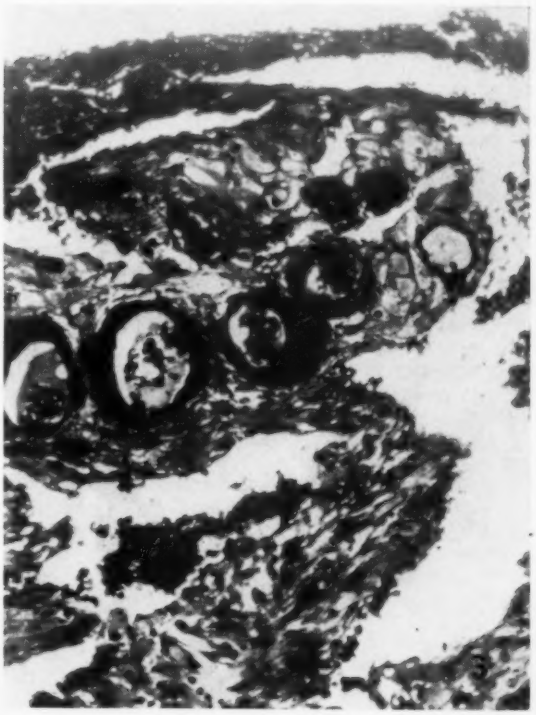

Fig. 3. See text.

By using the above method of obtaining specimens we have started a study on similar lines, and Fig. 1 shows a placenta from a 19-year-old primiparous Coloured patient who was admitted as an emergency case suffering from fulminating toxaemia at 30 weeks. A Caesarean section was performed within an hour of admission but the foetus was dead and a retroplacental clot was found underlying the infarcted area of the placenta. Fig. 2 shows myometrial

arcuate vessels with microscopic changes generally associated with the changes that occur in the kidneys and suprarenal glands in systemic hypertension. Fig. 3 shows the arteriolar changes in the decidua of this case. The significance of these vascular changes is at present not clear. It is thought that the myometrial vascular changes may be found in normal multiparous uteri, possibly resulting from the enormous shrinkage which must of necessity take place after parturition. A study is in progress to determine the consistency with which such changes may be found in normal uteri.

The above anatomical findings lead one to postulate that, if they are significant, and if it is accepted that the foetal chorion is completely dependent upon its maternal blood supply, the changes in the delivered placentae in premature separation of the placenta and toxaemia of pregnancy must then be due to alterations on the maternal side of the placenta.

Clinical evidence of the reduced blood-flow in the intervillous space in eclampsia and essential hypertension was produced by Browne and Veall[20] in 1953 and by Morris Osborn and Wright[21] in 1955.

The problem whether the reduction in utero-placental blood-flow is the cause of hypertension or its effect is as yet unsolved.

The findings by Dixon and Robertson[19] that the spiral arterioles in a normotensive patient with renal impairment show medial degeneration and intimal proliferation, if valid, indicate that hypertension may not be the prime factor in decidual degeneration.

In conclusion I should like to mention a suggestion made by Prof. J. T. Louw that, as far as can be ascertained, toxaemia of pregnancy has not been recorded as a complicating factor in chorio-epithelioma, in which condition, as we know, the trophoblastic elements predominate. The absence of decidua in this condition provokes the thought that changes in the decidua may one day provide a clue towards the solution of the riddle of the toxaemia of pregnancy.

SUMMARY

1. Certain concepts regarding the anatomy of the placental circulation are discussed.

2. Pathological lesions are demonstrated in the decidua from a case of fulminating toxaemia of pregnancy complicated by accidental haemorrhage.

3. The possible aetiological role of these lesions in the causation of placental infarction and accidental haemorrhage is mentioned.

4. The absence of decidua in chorion epithelioma is mentioned as a clue in the aetiology of toxaemia of pregnancy.

SAMEVATTING

Sommige van die huidige opvattings aangaande die anatomie en patologie van voortydige loslating van die plasenta word genoem met verwysing na die embriologiese ontwikkeling van die plasentale bloedsirkulasie.

As aanvaar word dat die plasenta geheel en al afhanklik is van die moederlike bloedstroom vir voeding, kan afgelei word dat infarksie en voortydige loslating van die plasenta waarskynlik veroorsaak word deur vertraging van die intervilleuse sirkulasie, as gevolg van ateroomagtige veranderinge in die desiduale arteriole.

Die afwesigheid van desidua by gevalle van chorio-epitelioom stimuleer die gedagte dat degenerasie van die desidua 'n belangrike rol mag speel by die etiologie van toksemie van swangerskap.

I wish to express my thanks to Prof. James T. Louw for his encouragement. Special thanks are due to Mr. N. D. Constantine, of the Department of Obstetrics and Gynaecology, for his kind cooperation and assistance in preparing the histological sections and for the microphotography.

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INTERSEXUAL CAUSES OF PRIMARY AMENORRHOEA*

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Patients with primary amenorrhoea usually find their way to the gynaecologist rather than the physician. In the USA many gynaecologists are in fact physicians or endocrinologists rather than surgeons but, since this is not the case in this country, I hope I may be excused for talking about a problem which is primarily an endocrinal one. The management of such a patient is frequently a matter for nice cooperation between specialists of different disciplines, which may or may not include any actual surgery, and the natural coordinator of these efforts is the endocrinologist.

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I will divide the intersexual causes of primary amenorrhoea into those (more common) with a vagina, in which one must resist the tendency to give oestrogens without more ado and then hope for the best; and those without a vagina, where the tendency has sometimes been to get rid of the patient to the plastic surgeon as quickly as possible.

With Vagina

Gonadal Dysgenesis ('*Ovarian Agenesis*')

There seems little doubt that the commonest cause of primary amenorrhoea seen at Groote Schuur Hospital is gonadal dysgenesis. I cannot quote exact figures, but when Dr. Muller was registrar in gynaecology we saw most of the

cases of primary amenorrhoea, and gonadal dysgenesis was outstandingly the most frequent.

The three prime features of this condition are external female appearance and body-form, internal female organs, but gonads represented by mere white streaks of tissue on the posterior aspect of the broad ligaments. Other outstanding features include shortness of stature in almost all cases (about 4 feet 8 inches when adult), and very often certain congenital anomalies, such as a webbed neck, asymmetrical face, 'shield-shaped' chest, multiple black moles, and coarctation of the aorta. Of course, no secondary sexual characters develop (although we have occasionally seen some breast growth) and the axillary and pubic hair may be scanty.

Since this condition is a variety of primary gonadal failure, the pituitary gland overacts and produces an excessive amount of gonadotropic hormone (FSH), which can be measured in the urine. Recently we have learnt that about 80% of these patients are actually genetic males*, which can be quite easily discerned by examination of the chromatin of the nuclei in stained smears of the buccal mucosa. This finding is, of course, most helpful in diagnosis and, in fact, renders a diagnosis possible at an early age if, for instance, it be suspected simply on the basis of shortness of stature. From the theoretical point of view it means that something went wrong very early in foetal development so that the gonadal tissue did not mature into testes. In the absence of foetal gonadal influence, female internal organs and female body-form developed in accordance with the known basic or neutral body-pattern.

The final diagnostic court of appeal is by laparotomy with inspection and section of the primitive gonads themselves. I have absolutely no doubt that, where the diagnosis is uncertain despite the investigations mentioned above, laparotomy is not only justified but necessary.

Management consists of explanation and encouragement, and oestrogens. We do not tell the patient about her genetic sex; but we do tell her what we can achieve with oestrogens, that we cannot add to her height, and that she will not have children. We warn her not to waste money on expensive and entirely useless injections or visits to persons of doubtful medical ethics, but we point out that many girls like herself are happily and satisfactorily married. Oestrogens we usually give simply as stilboestrol, between 0·5 and 2 mg. per day for 4 weeks, with a week's break and so on. Therapy must be continued for life. It is not good enough to produce one artificial menstrual period and then not see the patient again. Oestrogens will achieve growth of the nipples and breasts, more mature feminization of body contours, growth of the vagina and uterus, and in many patients increased energy and a feeling of well-being. They certainly look and feel 'more feminine'. Perhaps oestrogens tend to delay premature aging, atheroma and osteoporosis. Regular withdrawal bleeds are usually obtained.

At this juncture I should like to quote from a letter I recently received from an intelligent patient with gonadal dysgenesis: 'After my operation in 1955 I almost went to pieces. I wondered if it was worth while going on living if I was to be a neuter creature who would never have children.' She then says how much she appreciated our straightforward explanation of her condition, and goes on: 'Then there are the physical changes your treatment has brought about. Although I've never been a fan of Diana Dors, those extra inches certainly have made a great difference. Before 1955 I was too shy and self-conscious to appear in a bathing costume or to undress with other girls. Today I practically live in the sea during the summer. Although the periods I have are 'synthetic' and I fully realize this, it's amazing the difference it makes psychologically to be like other women'. Further, 'People who have not seen me for a long time often don't recognize me; they all say I have grown at least 8 years younger during the past few years. In the past few years I've changed from an awkward, self-conscious, self-centred introvert into a healthy, happy person who is outside as much as possible and thoroughly enjoys the company of people of both sexes—without the aid of testosterone!'

Women with Testes (Oestrogen-producing Testes)

This syndrome is not so common as gonadal dysgenesis, but is not rare—we have seen 4 cases within the past 2 years. Patients present in two main ways—either in childhood with hernia, in which case the surgeon is amazed when the odd structure he removed from the hernial sac is reported upon as testis—or as primary amenorrhoea, in which case the gonads are intra-abdominal.

The latter patient usually appears as a well-built, normally tall, female with good breast development. Two virtually diagnostic clinical features are the total absence of axillary hair and scanty pubic hair, together with lack of uterus. Then, most important, the chromatin pattern is male.

We look upon these patients as examples of male pseudo-hermaphrodites with complete sex reversal—their testes produce oestrogens rather than androgens and their hormonal abnormality allows female anatomical development and later breast development. (It is possible that it is the absence of androgen, rather than an actual excess of oestrogen, which is really responsible for the intersexual state since, as we saw in gonadal dysgenesis, a lack of male hormone leads to the 'neuter' or female type of development.) Such patients should, of course, continue to live as females, and may marry happily (their libido appears to be extremely healthy). We usually recommend removal of the gonads because of their liability to malignant change, (±10%), after which oestrogens are necessary to prevent menopausal symptoms!

Female Pseudo-hermaphrodites (Congenital Adrenal Hyperplasia)

It is extremely important to recognize these patients, since appropriate treatment may be highly successful (though it should really start soon after birth). Undiagnosed patients may be thought to be males with extreme hypospadias and undescended testes, or females with large clitoris and primary amenorrhoea who become masculinized and hairy at puberty. Actually they are genetic females (nuclear chromatin is female), with ovaries, whose adrenals are producing too much androgen. The final diagnostic feature is the presence of a great excess of 17-ketosteroids (and particularly pregnantriol) in the urine. They possess uterus and vagina, and in fact if the adrenal activity can be suppressed, they can develop breasts, menstruate normally and even bear children.

The treatment is with cortisone or an analogue, such as prednisone, which will successfully suppress the adrenal overactivity. The only surgical procedure which may be

* Recent work has shown abnormal chromosome numbers to exist in gonadal dysgenesis, so that the term 'genetic male' is not correctly applied here. (This matter is discussed in an Editorial article on page 743 of this issue.—*Editor.*)

necessary is reduction of the size of the hypertrophied clitoris. If, however, the patient has been brought up as male, it may be best to do nothing, certainly once male-type puberty has occurred.

True Hermaphrodites

These patients, whose gonads contain both ovarian and testicular tissue, are not as rare as you might think. Here we are really only interested in those with sufficiently feminine form to have been brought up as female and then to be seen for lack of menses. There is great variety in this form of intersexuality, but all have some kind of ambiguous sexual development and almost all have a uterus and vagina. One or both gonads may have descended into the labial region (in which case the relevant gonad must be either a testis or an ovotestis). The genetic sex may be male or, more commonly, female. There is a single perineal orifice, which may be shown by catheterization and radio-opaque dye studies to lead to a urogenital sinus which drains both a female genital tract and the urinary system.

Secondary sexual characters of either sex may develop with maturity. Breast growth and menstruation may occur, or the menstrual flow may be obstructed to produce a haematometria or haematocolpos, or appear as periodic 'bloody urine'. The person usually continues to assume the role in which he has been brought up. In some instances both male and female roles may be adopted in sexual relationships, provided the environment and circumstances of the moment call for such virtuosity.

Treatment in general consists of full investigation, with laparotomy if necessary, followed by such surgical, plastic or hormonal therapy as may be suited to furthering the patient's life in the sex to which he feels he belongs.

Male Pseudo-hermaphrodite (Intermediate Cases)

These patients have a phallus of variable size, and persistence of Mullerian-duct structures with a vagina into which the urethra opens—really a urogenital sinus. The scrotum is bifid and neither gonad may be descended, so that the patients may be mistakenly regarded as female. The gonads, of course, are testes, and the genetic sex male. If the diagnosis is not made early, the advent of a male type of puberty will come as a shock.

It may be right to continue to deal with some of these patients as females, but each one deserves careful investigation and individual consideration.

Without Vagina

Male Pseudo-hermaphrodites (some cases)

These patients, again, have a phallus of variable size, with extreme hypospadias, a bifid scrotum and, usually, undescended testes. There is no vagina, or only a rudimentary one.

Left alone, a male puberty will ensue, or the patient may become eunuchoidal if the gonads are incapable of producing male hormone. Catheterization and dye injection discloses no Mullerian-duct structures, the nuclear chromatin pattern is male, and gonadal biopsy reveals only testicular tissue.

Congenital Lack of Vagina

These patients appear normally female in all ways except that they lack a vagina and often uterus as well. Before an artificial vagina is constructed it is advisable to check that the nuclear sex is really female and that no Mullerian-duct structures open into the urethra. This is particularly important since the 'women-with-testes' syndrome or the true hermaphrodite may, rarely, lack a vagina—and the diagnosis in these instances would be completely missed if special investigations were not performed.

A lesser degree of abnormality, of course, is the incomplete canalization of the vagina—really an overgrowth of the labioscrotal folds, which may be combined with a rather large clitoris. Catheterization of the single perineal opening will readily separate these cases from those where no vagina is present at all.

CONCLUSION

Unless there is an obvious cause for primary amenorrhoea, such as tuberculous endometritis, all patients presenting with this complaint should be submitted to certain definitive investigations. These may include:

1. Examination under anaesthesia.
2. Catheterization of perineal orifice(s) with injection of radio-opaque dye. (Further examination by a urologist may be necessary.)
3. Nuclear sexing, by examination of buccal smear or other tissue.
4. Urinary output of 17-ketosteroids.
5. Urinary output of FSH.
6. Biopsy of accessible gonad.
7. Laparotomy, with biopsy of intra-abdominal gonad.
8. Psychological appraisal of the patient's own sexual orientation, which may be extremely useful in helping to decide upon the final lines of treatment.

I am pleased to acknowledge the value of my discussions with Dr. R. Hoffenberg, who has always worked with me on this subject.

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VAN DIE REDAKSIE : EDITORIAL

GINEKOLOGIESE EN OBSTETRIESE BYDRAES

Van 2–5 Maart 1959 het die Agste Interim Kongres van die Suid-Afrikaanse Vereniging van Verloskundiges en Ginekoloë in Bloemfontein plaasgevind. Weer eens het die hoë gehalte van die referate bewys gelewer van die uitstekende navorsingswerk, klinies sowel as akademies, wat in hierdie belangrike vertakking van medisyne in ons land verrig word.

Die Groep verloskundiges en ginekoloë vorm vandag seker een van die mees aktiewe Groepe in die Mediese Vereniging asook in die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika. Een van die doelstellinge van die Groep is om die gehalte van verloskunde en ginekologie wat gepraktiseer word, die opleiding van studente en vroedvroue, en die kraamfasiliteite wat bestaan, te probeer verbeter. Daar word teenswoordig in die groter sentra ondersoek ingestel na die oorsake van die moederlike sterftesyfer, aangesien hierdie syfer 'n goeie maatstaf is van die gehalte van verloskunde wat gepraktiseer word. Hierdie werk word egter bemoeilik deurdat geneeshere nie altyd op die doodsertifikaat invul dat daar 'n swangerskap teenwoordig was, of 'n onlangse bevalling plaasgevind het nie. 'n Beroep word dus op alle praktisyns gedoen om, waar 'n sterfgeval enigsins verband mag hê met swangerskap of geboorte, dit onder die bydraende oorsake op die doodsertifikaat te stipuleer.

Dat die Vereniging van Verloskundiges en Ginekoloë aktief is, word ook bewys deur die feit dat daar al agt interim kongresse gehou is, en dat die bywoning by al die kongresse goed was. Die kongres in Bloemfontein is bygewoon deur dertig verloskundiges uit 'n totale ledetal van ietwat meer as 'n honderd. Aangesien dit die eerste byeenkoms in Bloemfontein was, is dit vanselfsprekend dat daar 'n mate van huiwering bestaan het of die poging suksesvol sou wees. Hierdie onsekerheid is gou uitgewis deur die vriendelike ontvangs van die besoekers en die nadere kontak wat die kongresgangers met hulle gashere en medekongresgangers in 'n kleiner stad kon maak. Die waarde van so 'n kongres lê nie alleenlik in die bywoning van die voordragte nie, maar ook in die wisseling van gedagtes wat plaasvind.

Soos blyk uit die bydraes wat ons in hierdie uitgawe plaas, is die gehalte van die gelewerde bydraes op 'n hoë peil. Ongelukkig is 'n paar van die goeie referate nie vir publikasie op hierdie stadium beskikbaar nie aangesien hulle vir proefskrifte gebruik sal word. Hieronder was daar 'n bydrae deur dr. N. Walker van die Mediese Skool, van Durban oor die onderwerp 'Fetale nood—'n indikasie of 'n verskoning vir keisersnee'. In hierdie uiters stimulerende referaat word getoon dat indien die gewone tekens van fetale nood geïgnoreer word, die resultate wat betref fetale verliese geensins hoër is as wanneer dadelik handelend opgetree word nie; met ander woorde, baie keisersneë word waarskynlik onnodiglik gedoen juis omrede van hierdie indikasies. Hierdie belangrike werk is nog aan die gang en die finale gevolgtrekkinge mag ons sienswyse insake die belangrikheid van die tekens van fetale nood aansienlik verander.

Dr. S. Shippel van die Mediese Skool, Universiteit van die Witwatersrand, en een van ons uitstaande ginekologiese patoloë, het met sy oorspronklike navorsingswerk oor die tekasel van die ovarium wêreldberoemdheid verwerf. In die afgelope tyd het hy hom toegespits op die studie van die plasenta. Dit is ongelukkig dat sy bydrae oor die patologie van die ouderdomsverskynsels in die plasenta nie vir druk in hierdie uitgawe gereed is nie. Diegene wat in Bloemfontein na hom geluister het, sal dr. Shippel se geïnspireerde lesings nie gou vergeet nie. 'n Wetenskaplike wat so besiel is met sy werk moet altyd indrukwekkend wees, veral as hy ook onoortreflik as leermeester is.

Die kollegas van Bloemfontein het die kongres met graagte gereël. Met die oog op die beplanning van 'n nagraadse mediese skool help so 'n byeenkoms baie om die regte geestesstemming en atmosfeer onder kollegas sowel as onder die betrokke owerhede te skep. Met die snelle ontwikkeling wat in die Oranje-Vrystaat plaasvind en die geweldige toekoms wat die ontginning van goud en uraan beloof, is die dae waarin hierdie Provinsie as die aspoester van die Unie beskou is, haas aan die verbygaan. Die Nasionale Hospitaal in Bloemfontein beskik tans oor 670 beddens en met die aanbou van die nuwe hospitaal vir Naturelle sal dit vermeerder tot oor die 1,000 beddens. Die kern vir die skepping van 'n nagraadse skool bestaan dus en behoort ten volste gebruik en ontwikkel te word.

SECOND THOUGHTS ON SEX REVERSAL

The discovery that men and women can be distinguished by the pattern of their cell nuclei led to the belief that Turner's syndrome (gonadal-dysgenesis-with-female-body-form) occurred in a genetic male, while Klinefelter's syndrome, a form of hypogonadism with male body form, occurred in a genetic female. Thus the patients concerned appeared to be of a physical sex opposite to that of their gene structure, a state of affairs which became known as 'sex reversal'. This conception, however, has had a very short life, and has recently been destroyed by some interesting work on chromosomes which is summarized in three papers in the same issue of the *Lancet*.[1-3]

To get to this point we must go back a little in the study of chromosomes. In drosophila the study of chromosomes proved comparatively easy, since not only can the chromosome responsible for a particular feature be identified, but

even the exact portion of that chromosome where the relevant gene lies. In man, on the other hand, most of the assumptions concerning the physical basis of inheritance have been obtained by analogy with other species. Recent improvements in histological technique have enabled more direct observations of human chromosomes to be made during mitosis in tissue culture. Improvements in staining, the 'squash' method of making preparations, hypotonic solutions to 'expand' the cells, and the use of colchicine have all played a part. Colchicine has the property of halting the mitotic process halfway and also of inhibiting spindle formation, so that the individual chromosomes remain separated.

One of the first results of these methods was the demonstration that man has 46 chromosomes, and not 48, as had been believed for many years. Now, with the use of short-term cultures of sternal marrow, the authors writing in the *Lancet*[1-3] have demonstrated deviations from this chromosome number. These deviations have great clinical significance, e.g. cases of chromatin-positive (i.e. female-nuclear-pattern) Klinefelter's syndrome are found to have 47 chromosomes, while cases of chromatin-negative Turner's syndrome have only 45 chromosomes. The fact that two different teams of workers have made the same observations makes it probable that these numbers apply to all cases of the two conditions.

In each disease it appears that the abnormality lies in the sex chromosomes, although this is not completely certain, because the X and Y chromosomes cannot be identified from other similarly shaped chromosomes with absolute surety. It is a reasonable assumption, nevertheless, that the Klinefelter syndrome is characterized by the sex system XXY, and Turner's syndrome by XO. Some interesting conclusions can be drawn from this assumption. The sex chromatin mass which we have used for our clinical 'genetic sexing' certainly appears to represent the XX chromosome pair (i.e. the normal female apparatus). It tells us no more than that, however, and what we believed to be XX chromosomes in Klinefelter's syndrome were really XXY, while the assumed XY (chromatin-negative pattern) in Turner's syndrome was really XO. Consequently, the term 'sex reversal' becomes plainly incorrect, since the original genetic sex is indeterminate. Therefore, at the present time, we cannot say whether the XXY pattern indicates a *male* who has gained an X or a *female* who has gained a Y; or whether the XO (Turner's) represents a male who has lost his Y or a female who has lost an X. It makes us a little happier now in talking to patients with either of these conditions. If they should hear about their 'nuclear pattern', at least it can lead to no direct assumption of genetic sex'—we can with lighter heart inform them that their genetic sex does not oppose their apparent sex. Finally, these new discoveries appear to indicate the importance of the Y chromosome in man. In drosophila the Y has no importance with regard to sexual differentiation, so that an XXY is a normal female and an XO a normal male. In man, the Y chromosome, however, must surely have some active masculinizing function.

The third condition which has been investigated by modern methods is mongolism. In this disease, too, 47 chromosomes have been found, but the extra one is not a sex chromosome. Penrose and his co-workers[1] report a remarkable case of Klinefelter's syndrome and mongolism combined, in which there are two extra chromosomes, a total of 48. What will be the next condition to show chromosome abnormalities? We can be sure of advances in this field. What, for instance, of another variety of apparent 'sex reversal'—the normal-looking females with intra-abdominal oestrogen-producing testes, and chromatin-negative nuclear pattern? What of the true hermaphrodite, with mixed male and female sexual apparatus, whose chromatin pattern has been found to be either positive or negative? One thing we have surely learnt—the danger of introducing new nomenclature on an insecure basis.

1. Ford, C. E., Jones, K. W., Miller, O. J., Mittwoch, U., Penrose, L. S., Ridler, M. and Shapiro, A. (1959): Lancet, I, 709.
2. Jacobs, P., Baikie, A. G., Court Brown, W. M. and Strong, J. A. (1959): *Ibid.*, I, 710.
3. Ford, C. E., Jones, K. W., Polani, P. E., de Almeida, J. C. C. and Briggs, J. H. (1959): *Ibid.*, I, 711.

DIE GEVAAR VAN UTERUSRUPTUUR NA KEISERSNEE GEVOLG DEUR NORMALE KRAAM*

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Dit is vandag gebruiklik om in ongeveer 2-7% van alle bevallings 'n keisersnee-operasie uit te voer.[1-5] Die meeste van hierdie pasiënte is primigravidae. Die indikasies vir keisersnee is van 'n verbygaande aard (placenta praevia, abruptio placentae, toksemie, en uitgesakte naelstring, ens.) in ongeveer 50-60% van gevalle.[2, 6, 7]

Die probleem van die hantering van pasiënte gedurende swangerskap en kraam na die uitvoering van 'n keisersnee is reeds dikwels bespreek. Daar is diegene wat die houding inneem dat indien die pasiënt eenkeer 'n keisersnee gehad het, daar altyd weer 'n keisersnee uitgevoer moet word, en dan ook verkieslik 'n elektiewe keisersnee. Andere, weer, laat elke pasiënt wat 'n keisersnee gehad het voortgaan totdat sy weer in kraam kom en besluit dan of die keisersnee herhaal moet word of nie. Tussen hierdie twee groepe kry ons verskeie grade van mening, maar die mees algemene mening is seker dat indien die indikasie vir die eerste keisersnee van verbygaande aard was, dit redelik is om die pasiënt weer 'n kans te gee om normaal te kraam. Ons vind dus dat tussen 30 en 45% van pasiënte na 'n keisersnee vaginaal verlos word.[7-10] Dit is dan in hierdie groep van geslaagde verlossings na vorige keisersnee-operasies wat ons belangstel en die vraag wat ons nou wil stel is: Indien die pasiënt 'n normale verlossing na 'n keisersnee deurmaak, wat is die posisie met haar volgende kraam?

Geslaagde Verlossings

Daar is verskeie beskrywings van geslaagde verlossings by die tweede en daaropvolgende bevallings na keisersnee en die volgende syfers sal aandui wat moontlik is:

* Lesing gelewer tydens die Agste Tussentydse Kongres van die Suid-Afrikaanse Vereniging van Verloskundiges en Ginekoloë (M.V.S.A.), Bloemfontein, 2-5 Maart 1959.

TABEL I GESLAAGDE VAGINALE VERLOSSINGS NA KEISERSNEE

Aantal vaginale verlossings na keisersnee	*Aantal pasiënte*	
	Nattrass[11]	*Schmitz en Gajewski*[10]
2	12	15
3	6	7
4	1	4
5	1	1

Uterusruptuur

Bogenoemde syfers sluit nie, sover 'n mens van die artikels kan oordeel, enige gevalle van uterusruptuur in nie. Schmitz en Gajewski gaan sover as om hierdie feit te beklemtoon en te sê dat 'n suksesvolle vaginale verlossing na 'n keisersnee 'n aanduiding is daarvan dat daar baie min verdere gevaar vanweë ruptuur van die litteken vir daardie pasiënt voorlê. In verband met hierdie bewering word hulle egter vasgepen deur Eastman[7] wat uit die ervaring aan Johns Hopkins-Hospitaal konstateer dat 2 uit 10 gevalle van uterusruptuur wat na keisersnee plaasgevind het, die geskiedenis gegee het van 'n normale verlossing tussen die keisersnee en die uterusruptuur. Een van die pasiënte het 3 vaginale verlossings na die keisersnee gehad en toe, in haar 5e swangerskap, het die uterus een aand geruptuur terwyl sy 3 weke voor voltyd rustig die koerant gesit en lees het. Jackson[12] sê ook die volgende: ‚Repeated vaginal deliveries will also weaken the scar and it does not follow that because the patient has had one safe vaginal delivery, she can have several more'. En Marshall[13] beaam dit as volg: ‚Though there may have been vaginal deliveries following the section, the risk of rupture, though less, will still be there'.

Om dan die gevaar van uterusruptuur na 'n normale verlossing wat op keisersnee volg te beklemtoon, wil ek die volgende 3 gevalle beskryf:

Geval 1

Hierdie Blanke pasiënt het in 1951 op 21-jarige leeftyd 'n keisersnee gehad, skynbaar vir inersie van die uterus. Die keisersnee was van die laer segment tipe (Kerr). Die aard van die puerperium is ongelukkig nie aan my bekend nie. Die baba het 6 pd. 10 oz. geweeg. Aan die begin van 1956 is haar 2e kind normaal gebore (gewig 6 pd. 14 oz.). Op die eerste dag na die kraam het sy 'n ortopediese toestand ontwikkel wat gediagnoseer is as 'n septiese gewrigsontsteking van die regter iliosakrale gewrig. Geen spesifieke behandeling is hiervoor toegepas nie, maar na etlike maande het die toestand verbeter en kon sy weer heeltemal goed loop. Ek het haar vir die eerste maal op hierdie stadium gesien (2 maande na haar 2e kraam) toe sy met krukke geloop het, en daar by ginekologiese ondersoek niks abnormaals buiten 'n beweeglike eerstegraadse retroversie van die baarmoeder gevind is nie. Na 6 maande het sy weer swanger geword en haar baba ongeveer teen die middel van Mei 1957 verwag. Gedurende die swangerskap het sy o.a. gekla van pyn in die linkersy.

Kraam het begin om ongeveer agtuur die aand van 6 Mei en sy is om middernag in die kraaminrigting toegelaat. Aan die begin het die sametrekkings nie lank geduur nie en was nie besonder sterk nie. Baie kort na toelating is 'n rektale ondersoek gedoen en ontsluiting van net een vinger vasgestel. Die kind het in die R.O.L.-posisie gelê en die stand was 0. Die vliese was nie gebreek nie. Instruksies is gegee om haar noukeurig dop te hou weens die vorige keisersnee, en die kraam het heeltemal bevredigend gevorder tot ongeveer 2 vm. op 7 Mei. Op dié stadium het die suster gebel om te sê dat sy onrustig is omdat die fetale hart onverwags stadig geword het. Haar sametrekkings het kort voor hierdie tyd taamlik sterk geword en was baie pynlik, maar tussen die sametrekkings was daar goeie ontspanning van die uterus en die buik kon betas word sonder veel pyn vir die pasiënt. Haar pols was heeltemal normaal. Die teater is onmiddellik bespreek vir keisersnee en 100 mg. petidien en 1/75 gr. atropien is voorgeskryf. Voor dit egter gegee kon word, terwyl ek by haar staan, het sy met die volgende sametrekking 'n skree gegee omdat iets binne-in haar geskeur het en 'n massa het op die onderbuik uitgestaan. Die fetale hart het binne 2 minute verdwyn en buiten een of twee minder sterk sametrekkings van die uterus het die sametrekkings opgehou. Sy het 'n bietjie *per vaginam* gebloei en die pols het vinniger en swakker geword. Intraveneuse toediening van 5% dekstrose in soutoplossing is onmiddellik begin en bloed is vir haar bestel. By laparotomie was die kind nog in die heel vrugsak in die buikholte voor die uterus te vind en die plasenta was in die skeur van die litteken van die vorige keisersnee. Die hele wond het oopgeskeur, maar dit het dieper af gestrek aan die regterkant as aan die linkerkant. Daar was nie ernstige beskadiging van weefsel nie en aangesien ek daarvan bewus was dat die pasiënt baie angstig was om nog 'n kind te hê, het ek besluit om net die rante skoon te knip en die wond te heg. Twee pinte bloed is vir haar onder die operasie toegedien. Haar toestand by voltooiing van die operasie was heeltemal bevredigend. Ongelukkig is die kind nie geweeg nie, maar die indruk was beslis nie dat disproporsie in hierdie geval teenwoordig was nie. Die na-operatiewe verloop was heeltemal vlot. Die kwessie van verdere swangerskap is met die pasiënt bespreek en die risiko is baie duidelik aan haar gestel. Sy het egter besluit om daardie risiko te neem, en het in 1958 weer swanger geword. (Verwagte datum van bevalling, 3 November.) Haar swangerskap het heeltemal goed verloop, 'n elektiewe keisersnee is uitgevoer en 'n baba van ongeveer 6 pd. verlos. By hierdie operasie (uitgevoer deur 'n kollega van my aangesien sy nie in Pretoria kon kraam nie) was daar geen teken van die litteken van die vorige uterusruptuur te bespeur nie. Sy is tydens hierdie operasie ook gesteriliseer.

Geval 2

'n Bantoevrou is op 2 September 1957 toegelaat met die geskiedenis dat sy in 1951 'n keisersnee gehad het en in 1954 'n normale bevalling. Albei kinders was in goeie gesondheid. Die indikasie vir die vorige keisersnee was nie bekend nie. By toelating het sy die geskiedenis gegee dat die vliese reeds tevore gebreek het en by ondersoek was die ligging 'n R.O.A. met die kop beslis hoog. Die sametrekkings was nie besonder sterk nie en by vaginale ondersoek is vasgestel dat die os 2½ vinger ontsluit was. By hierdie ondersoek kon die promontorium van die sakrum gevoel word. Die pasiënt het daarna ongeveer 1½ uur lank taamlike sterk sametrekkings gekry, maar 3 uur na toelating was die fetale hart nie meer hoorbaar nie en die sametrekkings het opgehou. By betasting was die kop ingedaal maar by die gebied van die fundus was daar 'n abnormale uitbulting wat beskou is as die liggaam van die fetus. Met die diagnose van uterusruptuur is die buik oopgemaak en 'n subtotale histerektomie uitgevoer. Die vorige keisersnee was van 'n laer segment tipe en die wond was heeltemal oopgeskeur. Die skeur het afwaarts gestrek tot in die blaas. Die kop van die fetus was nog in die bekken vas toe die buik oopgemaak is. Die fetus was natuurlik dood. Die pasiënt het goed herstel.

Geval 3

Hierdie Bantoevrou is op 5 Augustus 1951 in kraam toegelaat as 'n para 4. Haar vorige obstetriese geskiedenis was soos volg:

1937 Normale bevalling. Kind na 18 dae dood.
1938 Keisersnee (? indikasie). Kind leef.
1941 Normale bevalling. Kind leef.
1946 Normale bevalling. Kind oorlede (20 maande).

Tydens die huidige swangerskap het sy geen voorgeboorte kliniek bygewoon nie en is in die afdeling toegelaat op die aand van 5 Augustus met die geskiedenis dat die kraam 'n dag en 'n half vantevore begin het. Haar vliese het 4 uur voor toelating gebreek. By toelating was sy in baring en in 'n geskokte toestand met 'n bloeddruk van 85/60 mm. Hg. en 'n polsspoed van 152 per minuut. Sy was anemies van voorkoms en het edeem van die vulva gehad. 'n Diagnose van uterusruptuur is gemaak en die pasiënt se toestand is deur middel van toediening van bloed en ander middels verbeter sodat 'n operasie uitgevoer kon word. Vier uur na toelating is sy in die teater onder narkose ondersoek en daar is bepaal dat die cervix vol ontsluit was met die baba in 'n stuitligging. Laparotomie is uitgevoer en 'n ruptuur van die ou keisersnee-litteken is gevind. Die kind was reeds dood en 'n subtotale histerektomie is uitgevoer. Na verdere bloedoortapping het die pasiënt bevredigend herstel buiten dat daar 'n mate van bekkenontsteking was wat na etlike dae gevolg het. Dit is ook met welslae behandel. Die gewig van hierdie kind was 7 pd. 4 oz. en by kliniese beraming by ondersoek onder narkose is die grootte van die bekken as redelik goed beskou.

Die voorkomssyfer van uterusruptuur na vorige klassieke

keisersnee is ongeveer 4%.[14, 24] Die voorkomssyfer van uterusruptuur na vorige laer segment keisersneë is in die omgewing van 0·65 tot 1·5%.[7,11,15-18] Die laaste syfer (1·5%) geld egter vir gevalle waar 4 of meer vorige keisersneë uitgevoer is en keisersnee dan herhaal is.[16] Bengtsson[6] beskryf 133 gevalle van vaginale verlossing na keisersnee waar geen uterusruptuur plaasgevind het nie.

Sover ek kon vasstel is daar geen syfers in verband met uterusruptuur spesifiek vir pasiënte wat 'n normale bevalling gehad het na 'n keisersnee en dan eers die ruptuur kry nie. Eastman[7] toon duidelik aan dat sulke gevalle wel voorkom, so ook die 3 gevalle wat ek beskryf het. Ons is dit dus heeltemal eens met sy woorde in hierdie verband: „I am certain that . . . optimism . . . in regard to the integrity of a section scar, just because the patient has had one or more intervening vaginal deliveries, is ill-founded."[7]

DIE HANTERING VAN 'N TWEEDE VERLOSSING NA KEISERSNEE

Die volgende punte is van belang by die hantering van 'n pasiënt wat 'n normale verlossing gehad het na 'n vorige keisersnee en dan weer 'n kraam tegemoet gaan.[8,10,11]

1. *Behandeling in 'n Inrigting*

Die pasiënt moet in 'n goed-toegeruste inrigting wees onder die sorg van 'n ervare verloskundige.

2. *Geskiedenis*

Die geskiedenis van alle vorige bevallings is belangrik by die oordeel aangaande die verloop van die kraam wat voorlê. Indien daar een of meer normale bevallings voor die uitvoering van die keisersnee was, sal dit gewoonlik beteken dat die pasiënt weer betreklik maklik behoort te kraam. Die vooruitsig is dus gunstiger as wat dit sou wees by die pasiënt wat geen vaginale verlossings voor haar keisersnee gehad het nie. Indien die pasiënt na haar keisersnee een of meer vaginale verlossings gehad het, is dit, soos ons nou net gesien het, tot 'n mate gerusstellend, maar 'n mens kan sekerlik nie geheel-en-al peil trek op die litteken ten spyte van die feit dat daar 'n normale vaginale verlossing tussenin was nie.

Wat betref die keisersnee self is dit van belang om te weet wat die indikasie vir die keisersnee was. Dit is vanselfsprekend dat die indikasie van 'n verbygaande aard moes gewees het omdat dit onder geen omstandighede gewaag kan word om 'n vaginale verlossing toe te laat wanneer daar byvoorbeeld uitgesproke disproporsie tevore teenwoordig was nie. Die tipe keisersnee wat uitgevoer is, is ook van belang. Die syfers wat reeds aangegee is, toon duidelik dat daar meer risiko vir uterusruptuur is wanneer 'n klassieke keisersnee uitgevoer is as in die geval van 'n snit van die laer segment. Daar is nogtans mense wat beweer dat toe Holland sy ondersoek ingestel het, daar nie antibiotika en die ander middels van behandeling was wat ons nou het nie, en dat hedendaagse syfers vir 'n ruptuur van 'n keisersnee-litteken laer is as in die dae van Holland. Die syfers wat genoem is, dui egter op 'n sterker litteken in die geval van 'n snit van die laer segment. Wat betref die sterkte van die twee tipes snit van die laer segment is daar nog nie enige deurslaggewende syfers gepubliseer nie. Die dwarssnit in die laer segment (Kerr) is 'n goed-beproefde snit en voorkeur word so dikwels hieraan gegee dat daar nie enige gepubliseerde reekse in verband met uterusruptuur na 'n lengtesnit in die laer segment (Krönig) is nie. Die gedagte is egter dat die lengtesnit in die laer segment beslis beter is as die klassieke snit.

Die laaste punt wat betref die vorige keisersnee is die aard van die puerperium. Indien daar uitgesproke sepsis teenwoordig was in die gebied van die uteruslitteken na die vorige keisersnee, moet daardie litteken in 'n sterk verdagte lig beskou word. Indien daar geen puerperale morbiditeit teenwoordig was nie, kan 'n mens met meer vertroue die volgende kraam tegemoetgaan.

3. *Die Cervix*

Ondersoek van die pasiënt aan die begin van kraam om die toestand van die cervix te bepaal is belangrik. Indien die cervix op daardie stadium mooi opgeneem is en reeds 'n paar vingers ontsluit is, is die vooruitsig vir die verloop van die kraam beter as wanneer die cervix dik en hard en nog lank is. Weer is dit net 'n algemene indruk in verband met die vooruitsig van die kraam, en kan 'n mens nie verseker wees dat met 'n gunstige cervix daar geen ruptuur van die vorige litteken sal plaasvind nie.

4. *Die Fetus*

Die grootte van die fetus is van belang in verband met die vooruitsig van die kraam. Die vorige geskiedenis van die grootte van die babas wat gebore is, sowel by die keisersnee as by die vaginale verlossings, moet verkry word. Hierdie feite asook die feit of hulle voltydse of vroeggebore kinders was, moet in ag geneem word in verband met die vooruitsig van die volgende verlossing. Ook moet 'n beraming gemaak word van die grootte van die fetus in die uterus, alhoewel dit baie moeilik is om 'n enigsins betroubare mening in die verband te vorm. Klem moet gelê word op die ondervinding by die Bantoe dat ons van tyd tot tyd by die multipara 'n keisersnee moet doen vir uitgesproke disproporsie. Dit is bekend dat die baba met agtereenvolgende bevallings groter word. Hierdie feit moet in gedagte gehou word in verband met die vooruitsig van 'n vaginale verlossing na 'n vorige keisersnee. Ander punte van belang in verband met die fetus is die ligging, die stand van die kop aan die begin van kraam, of die fetus lewe of dood is, en of daar enige fetale nood teenwoordig is.

5. *Die Litteken*

Die vraag of die litteken gaan hou of nie is heeltemal onvoorspelbaar. Soos aangedui in die punte wat nou net bespreek is, kan 'n mens nie in enige opsig seker wees dat omdat die tekens in 'n bepaalde geval gunstig is, daardie litteken sal hou nie. Een punt wat nie tevore bespreek is nie, is die feit dat by 'n vorige klassieke keisersnee daar 'n groter kans is dat 'n volledige ruptuur van die litteken met uitdrywing van die kind in die buikholte sal plaasvind, in teenstelling met die ondervinding by die laersegment-litteken dat 'n onvolledige ruptuur dikwels plaasvind en dat die fetus nie altyd deur die ruptuur in die buikholte uitgedryf word nie. Om die rede is daar by ruptuur van die laer segment 'n groter kans dat 'n lewende baba gekry kan word selfs na die wond padgee. 'n Ander aspek in verband met die litteken wat interessant is, is dat indien dit gaan ruptuur dit nie noodwendig sal uithou tot in kraam nie. Dit gebeur selde dat 'n litteken van die laer segment voor kraam padgee, maar dit kan definitief gebeur. Hierdie punt word as 'n argument aangevoer ten gunste van elektiewe keisersnee ongeveer 2 weke voor voltyd by alle pasiënte wat 'n vorige keisersnee gehad het. Ons stem nie met hierdie sienswyse saam nie, maar die argument is wel geldig.

Laastens is daar die vraag oor hoe dikwels daar 'n waarskuwing is van dreigende uterusruptuur voor die litteken padgee. Dit is verbasend hoeveel gevalle van uterusruptuur plaasvind sonder dat daar 'n uitgesproke waarskuwing is van 'n dreigende uterusruptuur—in ten minste 40% van gevalle is dit die geval.[19] Daar is selfs in sommige gevalle weinige tekens na die ruptuur plaasgevind het. Dit geld gewoonlik vir 'n gedeeltelike ruptuur van 'n litteken van die laer segment. Die aard van sametrekkings en die duur van kraam sal noodwendig 'n invloed hê op die vraag hoe lank die litteken in kraam sal kan hou voor dit skeur.

6. *Die Statistieke*

Die besluit on 'n vaginale verlossing toe te laat of om 'n keisersnee te doen, moet geneem word in die lig van die volgende oorwegings:

(*a*) Ruptuur van die litteken vind net in ongeveer 1% van gevalle plaas wat 'n vorige laersegment keisersnee gehad het.

(*b*) Met die uitsondering van sekere uitstaande reekse[25,26] is die perinatale sterftesyfer vir keisersnee-babas meer as 1% hoër as by normale verlossing *per vaginam*, selfs by elektiewe keisersnee.[27,28]

Solank hierdie syfers geldig bly, is dit dus nie geregverdig om in gevalle wat 'n vorige laersegment keisersnee gehad het, gevolg deur normale kraam, roetinegewys 'n elektiewe keisersnee te doen nie.

(*c*) Die syfer van 4% vir ruptuur na die klassieke tipe keisersnee sou wel 'n roetine elektiewe keisersnee daarna regverdig, maar dit is juis in hierdie gevalle dat die ruptuur dikwels heelwat voor die aanvang van baring plaasvind.

SWANGERSKAP NA UTERUSRUPTUUR

Na aanleiding van die eerste geval wat ek beskryf het, het ek hierdie saak nagegaan. Daar word in die literatuur baie min melding van swangerskap na uterusruptuur gemaak en net 4 gevalle is in die huidige reeks artikels gevind. Die eerste geval van Pettit[20] het 'n klassieke keisersnee gehad, daarna 'n uterusruptuur waarby haar toestand bedenklik was, maar sy het herstel, weer swanger geword en weer 'n gedeeltelike uterusruptuur gehad. Die baba is egter gelukkig gered en sy het goed herstel.

Die tweede geval word deur Lyon[21] beskryf en toon ooreenstemming met die benarde soort obstetrie wat ons dikwels ook onder die Bantoe moet uitvoer. Lyon se pasiënt het 'n uitgesproke vernoude bekken gehad en haar eerste 2 swangerskappe het op vernietigende operasies na die dood van die fetus uitgeloop. By haar derde kraam is sy in 'n geskokte toestand toegelaat nadat pogings tot verlossing buite die hospitaal gemaak is. Daar was 'n skeur van die perineum, die linkerkant van die vagina, die cervix en tot in die breë ligament, maar nie deur die peritoneum nie. Die kind is weer deur 'n vernietigende operasie verwyder. By haar volgende kraam het sy agt uur na die begin van baring na die hospitaal gekom met 'n uterusruptuur aan die linkerkant van die laer segment. Die kind is gelukkig lewendig uitgehaal deur 'n klassieke keisersnee en 'n subtotale histerektomie is uitgevoer.

Dewhurst en Rowley[22] beskryf 'n geval wat haar eerste kind verloor het met 'n tangverlossing, die tweede bevalling was normaal, 'n keisersnee is gedoen by die derde, daar was 'n ruptuur van 2½ duim breedte aan die linkerkant van die vorige keisersnee-litteken by haar vierde (kind lewendig), en na herstel van hierdie gedeeltelike ruptuur het sy weer swanger geword en 'n elektiewe keisersnee ondergaan gevolg deur sterilisasie.

Eastman[23] noem 'n geval van dr. W. Drummond Eaton wat met haar eerste kind 'n laer segment keisersnee gehad het, 'n uterusruptuur deur die litteken by haar tweede bevalling, maar sy het daarna weer swanger geword en 'n elektiewe keisersnee deurgemaak, met 'n lewende baba. Met reg was die kommentaar hierop: „It requires quite a little fortitude to pursue this course, but information of this subject is so meager at present that there is no way of saying just how much hazard is involved.' Dit wil voorkom dat, indien die ruptuur van 'n keisersnee-litteken nie volledig is nie, of as die rante na volledige ruptuur mooi skoon is en die hegting goed, die risiko van 'n redelike aard is en die kanse op uterusruptuur na dié tyd nie veel groter as by 'n gewone herhaal-keisersnee nie.

SUMMARY

The occurrence of one or more vaginal deliveries after previous Caesarean section does not mean that the danger of rupture of the scar can be excluded in subsequent pregnancy or labour. The scar of a previous lower-segment operation is less likely to rupture than that of a previous classical section.

Three cases of uterine rupture are described in which the patients had had normal confinements following previous Caesarean sections, two of which were of the lower-segment type.

The management of a second delivery following Caesarean section is discussed.

Finally, pregnancy following uterine rupture is discussed in view of the fact that the first case described had a subsequent successful pregnancy, terminated by elective Caesarian section and sterilization.

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CERTAIN ASPECTS IN THE APPROACH TO HYPERTENSION IN PREGNANCY*

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Platt[1] stated: 'Hypertension is something like obesity, something in which there are no limits, and where you cannot point to people and say, these are normal and those are abnormal.' For the obstetrician a clear concept of what is to be considered as normal blood pressure in pregnancy is imperative because of the dangers that even minimal abnormalities may entail for the mother and her baby. Is the generally recognized upper limit of 140/90 mm. Hg on 2 or more occasions acceptable to us as the level whereby all patients can be assessed to be hypertensive or not? Can we, with only this as basis, predict whether the complications of hypertension in pregnancy will ensue or not? If not, then the approach to this, the most acute problem in modern obstetrics, will have to be reconsidered. There are many authorities who for a long time have doubted whether blood pressure readings lower than 140/90 mm. Hg ought not to be regarded as significant of hypertension. For 30 years F. J. Browne[2, 3] has taught that any elevation of the blood pressure above 120 mm. Hg systolic and/or 80 mm. Hg diastolic should be regarded as suspicious, and transient rises in the systolic, diastolic or both readings, even in response to excitement, should be regarded as significant. These patients he classified as the 'labile' blood-pressure group. Morris and McClure Browne[4] consider these 'no-man's-land' cases as the troublesome and unpredictable ones; they found that 39% of them ultimately develop pre-eclamptic toxaemia.

The evaluation of hypertension in a pregnant patient may be of great importance since treatment hinges upon the assessment. There are many factors that may influence blood-pressure readings. The importance to be attached to them varies considerably; it often seems to be clouded by personal bias. The reasons for this unsatisfactory state of affairs may be briefly enumerated as follows:

1. *The duration of pregnancy.* Reid and Teel[5] and Chesley and Annitto[6] showed that the majority of hypertensives, like normotensives, have a drop in blood pressure at mid-pregnancy. Difficulty may be encountered when a patient is seen for the first time in the middle trimester of pregnancy. She may present with a normal blood pressure, whereas this mid-pregnancy drop may be masking a higher blood-pressure level.

2. *Individual variability* from day to day and hour to hour, and in response to excitement. This must influence a number of blood-pressure readings, perhaps especially in pregnant women.

3. *The range in the circumference of the arm.* This may be as great as 15–50 cm., caused either by fat or oedema. Fraser Roberts[7] found a rise in blood-pressure readings of 5 mm.

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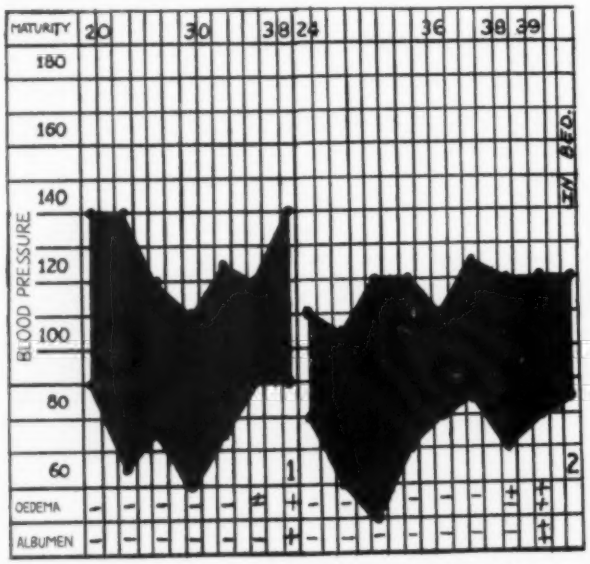

Fig. 1. Between the 28th and the 32nd weeks this patient's blood pressure was completely normal. The ultimate development of hypertension+proteinuria and oedema could only have been expected if the hypertensive blood pressure reading at the 20th week was known.

Fig. 2. Although this patient's blood pressure readings stayed within the recognized normal limits of 140/90 mm. Hg, she developed eclampsia while in bed and well sedated.

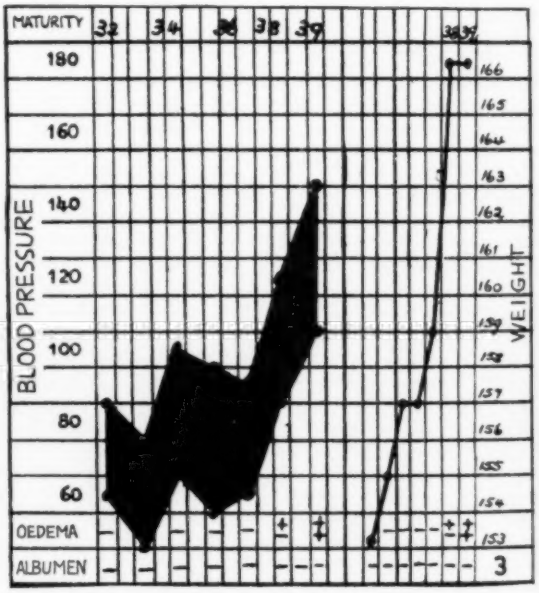

Fig. 3. The sudden rise in blood pressure from 95/55 mm. Hg at 37 weeks to 125/90 at 38 weeks should have been the warning sign, especially as it was associated with a marked increase in weight. At the 39th week she was truly hypertensive and was admitted with a mixed type of accidental haemorrhage.

Hg systolic and 3 mm. Hg diastolic per 4 cm. increase in arm circumference.

4. *The taking and interpreting of the blood pressure* may vary in different observers. The diastolic pressure may be recorded as the reading at which there is a change in the sounds, or when the sounds disappear. This readily accounts for a difference of 5–10 mm. Hg or more.

5. *A low blood pressure at the beginning of pregnancy.* What is the significance of a rise of the blood pressure during pregnancy from an initially normal or low reading to one still within the recognized upper limit of 140/90 mm. Hg? Should a pregnant woman who at the beginning of pregnancy has a blood pressure of 110/60 mm. Hg and at 38 weeks one of 135/85 not be considered as much hypertensive as the patient who has a blood pressure rise from 125/75 to 150/100? In each case the rise is 25 mm. systolic and 25 mm. diastolic. One is tempted to regard the findings in the first group as significant, for experience shows that many patients in this group will probably develop severe complications of hypertension, especially when the rise is a rapid one. It is sometimes associated with other signs of toxaemia such as oedema or proteinuria.

6. Difficulty is experienced in pinning a diagnostic label to the patient who has a blood pressure of 135/80 mm. Hg, with or without oedema, and no proteinuria or only a trace, who after 6–12 hours of labour shows a rise in blood pressure to 160/110 mm. Hg and an increase in proteinuria. When did she become hypertensive?

The obvious conclusion is that there is no blood-pressure level at which all patients can be diagnosed as being hypertensive. No sure prediction can be made whether a patient will be prone to the complications of hypertension. Each case, therefore, must be assessed on its own merits. The significance attached to variations must be determined by associated findings, viz.: (1) age and parity, (2) history of previous hypertensive episodes, (3) the rise in blood pressure and the rapidity with which it occurred, from the pre-pregnancy or early pregnancy state, and (4) the presence of associated signs of oedema and/or proteinuria.

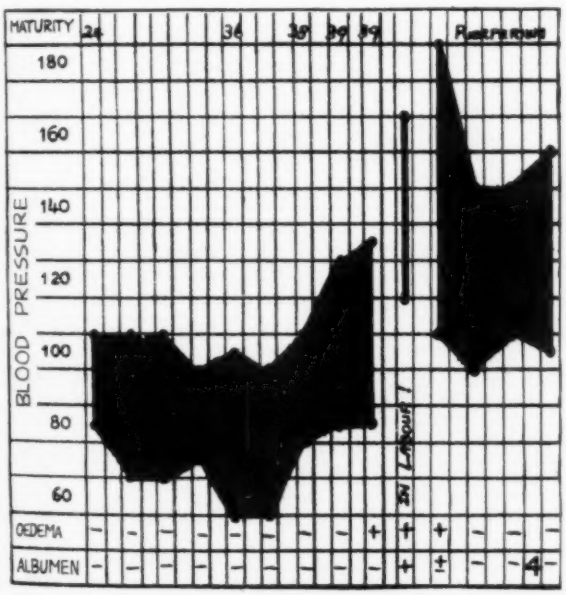

Fig. 4. The day before admission, this patient's blood pressure was 135/85 mm. Hg with slight oedema present. Not only in labour did her blood pressure rise to 170/120 but in the puerperium she stayed hypertensive and had to be sedated heavily.

USE OF HYPOTENSIVE DRUGS

An analysis of what happened to the hypertensives (the standard of hypertension being a blood pressure of 140/90 mm. Hg or more) in our unit during the years 1957 and 1958 is shown in Table I. These figures emphasize that hypertension and its associated complications constitute the major problems in modern obstetrics.

TABLE I. HYPERTENSIVES ADMITTED IN 1957 AND 1958

Number of patients admitted	14,834
Incidence of hypertension	11·2%
Incidence of eclampsia in hypertensives	4·1%
Incidence of accidental haemorrhage in hypertensives	8%
Foetal loss in hypertensives (with and without proteinuria)	12·4%

Of the maternal deaths 46·6% were due to hypertensive (+ proteinuria) complications.

In view of these facts and the acute shortage of antenatal beds, a new approach to the management of hypertensives was (and still is) being tried in our unit, viz. the use of certain hypotensive drugs. The aims are obvious:

1. To treat as many of these patients as out-patients as is possible. Not only would this ease the antenatal bed position but would relieve the domestic, social and economic problems of the patient, brought about by long sojourn in hospital.

2. To prevent the development of proteinuria and oedema.

3. Should oedema or proteinuria have developed, it was hoped to reverse the condition or at least to tide the patient over to possible viability of the foetus; that is to say, to obviate prematurity with its high foetal mortality.

The drugs used were mevasine in combination with chlotride, and serpasil in a few cases. The routine followed was as follows:

1. An assessment was made whether the patient was suitable for treatment. Advanced retinopathy, severe albuminuria and cardiac failure (associated with hypertension) were regarded as contra-indications for continuation of pregnancy.

2. In the cases regarded as suitable for treatment, the minimum dose of 2·5 mg. of mevasine t.d.s. and 1,000 mg. of chlotride on alternate days was given to the patient, either in hospital or as an out-patient, according to the severity of the hypertension and the presence of associated toxaemic signs. The dose of mevasine was gradually increased if necessary, but the maximum of 10 mg. t.d.s. was never exceeded. If the blood pressure was still not controlled, serpasil was added to the treatment, either parentally (1 mg. every 3–4 hours) or orally (0·25 mg. 3 or 4 times a day). No response to treatment was taken as an indication for termination of the pregnancy.

3. If at all possible, mevasine treatment was stopped 3–4 days before any contemplated operative procedures, in view of the dangers of paralytic ileus in the mother or child.

4. During mevasine treatment, laxatives were given regu-

larly because of the severe constipation that is often found in association with the administration of this drug.

The results of this treatment in 169 cases are shown in Tables II, III and IV, with comments. Figs. 5 and 6 show the response of two of the patients treated.

TABLE II. TREATED HYPERTENSIVES: BEFORE 28 WEEKS

	Severer Group 160/100+		*Mild and Moderate Group*	
No. of patients	25		40	
Maternal mortality	0		0	
Foetal loss: Abortions	4		1	
Hysterotomies	2	= 38·5%	0	= 7·5%
Stillbirths	2		0	
Neonatal deaths	2		2	
No. of eclamptics	0		0	
No. of accidental haemorrhages	2	= 8%	0	
No. who developed proteinuria	6	= 24%	5	= 12·5%
Average weight of babies	5 lb. 6 oz.		7 lb. 4 oz.	
Average stay in hospital	19½ days		8½ days	

(*a*) The foetal loss of 38·5% is still very high in the severe hypertensives, although it should be noted that most of these patients were originally referred for termination of pregnancy. That of 7·5% in the mild and moderately severe hypertensives compares favourably with results claimed by workers like Taylor, Tillman and Blanchard.[9]

(*b*) Proteinuria developed in twice as many cases in the severer hypertensives as in the mild and moderate ones.

(*c*) The average weight of the babies in the severer group is much less than in the mild and moderate group despite the fact that the average stay in hospital is twice as long in the former.

(*d*) The incidence of accidental haemorrhage (8% in the severer group) is not decreased by hypotensive drug therapy.

TABLE III. TREATED HYPERTENSIVES: AFTER 28 WEEKS

	Hypertensive only	*Hypertension + Proteinuria*
No. of patients	22	52
Maternal mortality	0	1 = 1·9%
Foetal loss: Stillbirths	0	4 = 11·5%
Neonatal deaths	0	2
No. of eclamptics	0	1 = 1·9%
No. of accidental haemorrhages	0	4 = 7·6%
Average weight of babies	7 lb. 2 oz.	7 lb.
Average stay in hospital	6 days	13 days

(*a*) It is obvious that the uncomplicated hypertension in pregnancy offers no great hazards to mother and child, but once proteinuria develops the outlook for both deteriorates.

(b) Again the incidence of accidental haemorrhage is 7·5%.

TABLE IV. TREATED 'NO-MAN'S-LAND' CASES ('LABILE' BLOOD-PRESSURE GROUP)

No. of patients	30
No. who developed hypertension	2 = 6·6%
No. who developed hypertension + proteinuria	3 = 10%
Maternal mortality	0
Foetal mortality	0
Average weight of babies	7 lb. 12 oz.
Average stay in hospital	6½ days

This series of cases is too small for any conclusions to be drawn from it, but it is perhaps with timely treatment in this group that the real value of hypotensive and diuretic treatment will be found. Further statistics will be published later.

PROGNOSIS

An important aspect to be considered is the prognosis of patients with hypertension in whom pregnancy has been allowed to continue. Our own hypertensive follow-up clinic was only started 1½ years ago and no analysis can as yet be made. Gladys Dodds[10] followed up hypertensives in subsequent pregnancies and came to the following conclusions:

(*a*) In patients who were over 30 years of age when the hypertension in pregnancy first manifested itself, an exacerbation of the hypertension in subsequent pregnancies was more likely to occur, proteinuria developed more frequently, and the foetal loss was increased.

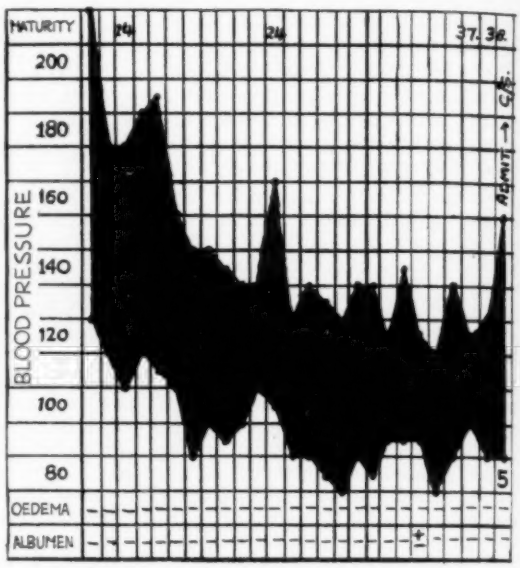

Fig. 5. This patient had a hysterotomy performed for hypertension in 1955 and a therapeutic abortion in 1957. As a known hypertensive she was referred to the gynaecological outpatients department in 1958 for termination of pregnancy and sterilization. After an initial admission to hospital for 5 days so that she could be fully investigated and treatment commenced, she was treated as an out-patient until she was admitted for Caesarean section. The weight of her baby was 5 lb. 13 oz. Both mother and baby were discharged fit after 10 days in hospital. At a subsequent visit at the hypertensive follow-up clinic her blood-pressure reading was 180/110 mm. Hg. She had been off treatment since the birth of her baby.

(*b*) Marked increase in the hypertension *did not* occur within the first 5 years. The hypertensive should therefore have her next baby as soon as possible.

(*c*) The higher the initial blood pressure with the previous pregnancy, the greater the risk of exacerbation of the hypertension and the development of proteinuria.

For the remote prognosis, it is best to refer to the results of the following workers:

1. F. J. Browne,[11] who stated that:

(*a*) 20–50% of hypertensives during pregnancy will develop chronic hypertension, depending upon whether the normal blood-pressure reading is taken to be 140/90 or 120/80 mm. Hg, whether there is a family history of hypertension, and the length of time that the hypertension lasted before delivery of the baby.

(*b*) Pre-eclamptic toxaemia will supervene in 20% of hypertensives.

(*c*) Chronic nephritis, if it ever occurs, must be an exceedingly rare sequel of pre-eclamptic toxaemia.

2. Poul Bechgaard, Andreassen and Hertel,[12] who, in following up 383 hypertensives for a 10–20 year period, found that:

(*a*) 57% of hypertensives during pregnancy were severely

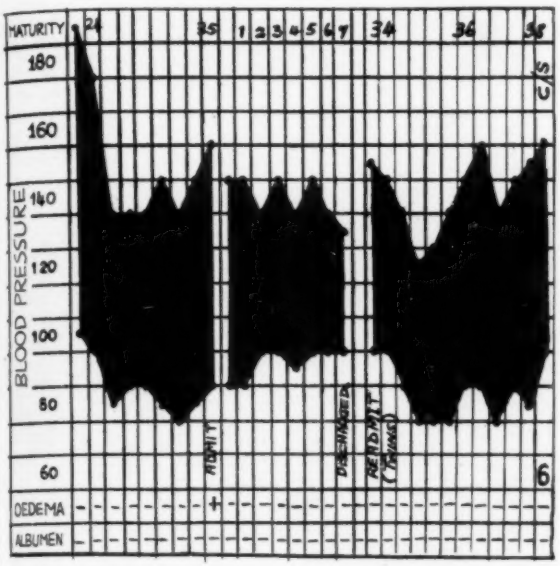

Fig. 6. This patient was a known hypertensive for years and was under constant treatment. She had no live children, despite this being her 3rd pregnancy. Her first pregnancy was of twins. Both children died neonatally as prematures, labour having been brought on for hypertension and proteinuria. In this last pregnancy her first admission to hospital was necessitated by the rise in blood pressure and the presence of oedema. The second admission was for the necessary rest when a twin pregnancy was diagnosed. At 38 weeks a Caesarean section was performed. Both babies were alive and were discharged after a 3 weeks' stay in hospital. The mother made an uneventful recovery. At a subsequent visit at the hypertensive follow-up clinic her blood-pressure reading was 190/115 mm. Hg. She had been off treatment since the birth of her babies.

hypertensive after the age of 45 years. In a control series of normotensives 21% became severely hypertensive. Before 45 years only 18% were severely hypertensive.

(*b*) Cardiovascular disease developed in 27% of hypertensives, renal disease in 2%.

(*c*) The mortality amongst those who were hypertensives because of pre-eclamptic toxaemia was much higher than amongst the essential hypertensives. The general health of the former was also much poorer.

CONCLUSIONS

1. The assessment of a pregnant patient for the condition of hypertension cannot be based on a blood-pressure level of 140/90 mm. Hg alone, but should be governed by many other factors.

2. Hypotensive drug therapy has a practical value in the treatment of hypertension in pregnancy. These patients can be treated as out-patients, with only the minimal stay in hospital. It also offers hope to some of the unfortunate women with severe hypertension and no live children.

3. The pregnancy associated with uncomplicated hypertension of the mild and moderately severe types does not constitute a severe obstetrical hazard. Even the more severe hypertensives can be controlled well enough not to endanger the life of the mother unduly, although the foetal loss is very high and the incidence of accidental haemorrhage is still about 8%.

4. Once proteinuria is superimposed, the mother's life is more endangered and the outlook for the baby deteriorates.

SAMEVATTING

Die begrip van wat met hipertensie gedurende swangerskap bedoel word is deeglik bespreek, en daar is tot die gevolgtrekking gekom dat die algemeen aanvaarde bloeddruklesing van 140/90 mm. Hg as basis om hipertensie te diagnoseer nie voldoende is nie. Ander faktore moet ook in ag geneem word.

Die waarde van hipotensiewe behandeling met mevasine, chlotride en serpasil in 169 gevalle is nagegaan, en die resultate behaal, uiteengesit.

I wish to express my thanks to Professor James T. Louw, Head of the Department of Obstetrics and Gynaecology, University of Cape Town, for his constant encouragement and helpful criticism, and the Superintendents of the hospitals concerned for permitting the analysis of these cases.

Spesiale dank aan my mede-kollegas vir die verwysing van sommige gevalle, asook die verplegende staf vir samewerking en troue versorging van hierdie pasiënte.

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DIE ESTROGEEN PRODUSERENDE TESTES SINDROOM*

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Die normale geslagsontwikkeling van die indiwidu tot man of vrou word bepaal deur (*a*) genetiese aanleg, (*b*) gonadale struktuur, (*c*) hormonale patroon, (*d*) primêre en sekondêre geslagsorgane en kenmerke, en (*e*) psigiese patroon. Hierdie ontwikkeling kan op verskillende maniere afwyk van die normale patroon en lei tot intersekse wat in die volgende vorms kan voorkom:

1. Ware interseks.

2. (*a*) Vroulike interseks met aangebore hiperplasie van die bynier. (*b*) Vroulike interseks sonder hiperplasie van die bynier.

3. (*a*) Manlike interseks met manlike of onsekere uitwendige geslagsapparaat. (*b*) Manlike interseks met vroulike

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uitwendige geslagsapparaat: (i) Die estrogeen produserende testes sindroom, en (ii) gonadale disgenese.

DIE ESTROGEEN PRODUSERENDE TESTES SINDROOM

Hierdie sindroom, wat ook bekend is as testikulêre tubulêre adenoom, testikulêre vervrouliking, Goldberg-Maxwell-sindroom, of, meer omskrywend, as die sindroom van testikulêre vervrouliking in manlike intersekse, kom waarskynlik meer dikwels voor as wat uit die literatuur blyk.

In 1953 kon Morris[1] slegs 82 verwysings na tipiese gevalle in die literatuur vind, waarby hy 2 van sy eie gevalle gevoeg het. In die Duitse literatuur is hierdie toestand egter meer algemeen beskryf en Prader[2] rapporteer 4 persoonlike gevalle en 2 ander wat binne 3 jaar in Zürich gediagnoseer is. Hy verwys ook na Wiedeman in Duitsland na wie in een jaar 15 gevalle verwys is vir chromatiengeslagsbepaling. Neem 'n mens verder in ag dat 10% van liesbreuke by kinders in dogters voorkom, is dit feitlik seker dat baie gevalle van die sindroom hier gevind sal word as roetine biopsies geneem word by breuk-hersteloperasies.

Nege gevalle[3-11] is beskryf in die Engelstalige literatuur na die publikasie van Morris[1] in 1953. In Pretoria is 2 tipiese gevalle in die afgelope 3 jaar gediagnoseer.

Die estrogeen produserende testes sindroom presenteer in skynbaar normale vroue met 'n manlike chromatienstruktuur, geheel of gedeeltelik onafgedaalde testes in die plek van ovaria, en geen uterus nie. 'n Duidelike erflike verband dui op 'n genetiese basis vir die wanontwikkeling.

Patogenese

Die voor die hand liggende patogenese is dat die testes die ontwikkeling van die Müllerse buise verhoed of teenhou by die stadium van geslagsdifferensiasie van die 7e tot die 9e week (17-33 mm. embrio). Hierdie potensialiteit van die testes is reeds in 1947 deur Jost bewys, maar of dit geskied by wyse van hormoonwerking of deur 'n ander onbekende organiseerder wat afgeskei word deur die testes, is nie bekend nie.

Dat die 9e week die keerpunt (*Drehpunkt*) is waar interseksuele ontwikkeling begin, blyk uit die ongeremde ontwikkeling van die vroulike uitwendige geslagsapparaat na hierdie tydstip. Verder besit hierdie testes nie spermatogonia nie, terwyl hierdie selle reeds by die normale embrio van 31 mm.[12] aangetref word.

Die opheffing van testikulêre vermanliking na die keerpunt kan te wyte wees aan 'n primêre testikulêre endokrienafwyking of 'n gebrekkige reaktiwiteit van die eind-orgaan. Die oorerflikheid van hierdie toestand dui op 'n genetiese faktor en die feit dat dit bewys is dat hierdie testes estrogeen of estrogene stowwe produseer, bevestig die hipotese dat 'n testikulêre afwyking die oorsprong van die toestand is. By die puberteit ontwikkel dan ook, i.p.v. manlike sekondêre geslagskenmerke, feitlik normale mammae en behalwe vir haar amenoree is die persoon 'n normale volwasse vrou.

Die chromatienpatroon is altyd manlik.

Simptomatologie

Goldberg en Maxwell[12] was die eerste om hierdie sindroom as 'n manlike interseks met volkome vervrouliking uit te ken. Die eerste geval wat beskryf is, is egter waarskynlik dié van Steglehner in 1817.[13]

Die uitstaande kenmerke van die sindroom, soos deur vorige werkers beskryf, word goed weerspieël in die gevalle van die 2 jong Bantoevroue wat hier beskryf word:

1. *Vroulike habitus* (Afb. 1). Albei pasiënte het innemende vroulike geaardhede getoon, hulle libido was heteroseksueel en daar was geen homoseksuele neigings by enige van die pasiënte nie. Die een Bantoevrou was getroud en die ander een gaan trou by haar ontslag uit die hospitaal. Tipiese ginekoïde bekkens het 'n lae voorkoms by Bantoes, en geen geslagsdifferensiasie van die bekkenvorms van die 2 pasiënte kon afgelei word nie. By hierdie gevalle was geen eunugoïde ontwikkeling te bespeur soos soms beskryf word in die sindroom nie.

Afb. 1. Pasiënt met testikulêre vervrouliking. Geringe pubiese beharing en geen okselbeharing nie. Normale borste, hipertrofiese klitoris en 'n inguinale regter-testis.

2. *Vroulike mammae* (Afb. 1). Normale vroulike borste was aanwesig en goed ontwikkel. Die tepels was klein by albei pasiënte.

3. *Beharing van die pubis en oksel* het in albei gevalle ontbreek en slegs die een pasiënt het geringe pubiese beharing getoon. In albei gevalle was die haarstruktuur fyn en sag met vroulike distribusie van die beharing van die kop.

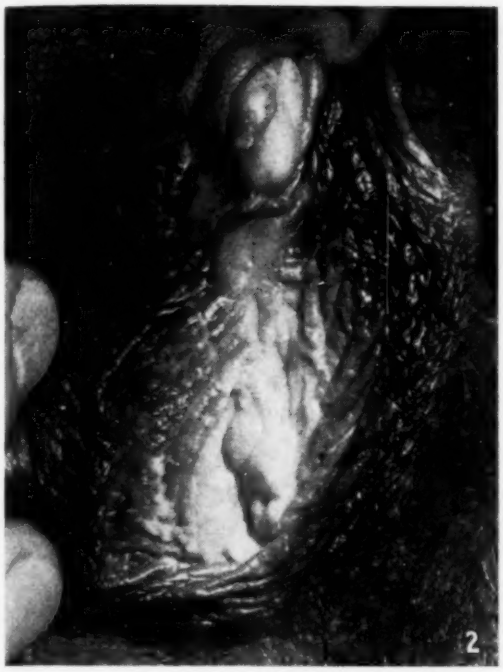
4. *Vroulike uitwendige geslagsorgane.* Geringe vergroting van die klitoris was aanwesig by die een pasiënt (Afb. 2). Hierdie bevinding is in teenstelling tot die meeste gepubliseerde gevalle waar die klitoris as klein of normaal gerapporteer is. Die labia by albei gevalle was normaal met geen onderontwikkeling van die labia minora soos by baie van die gepubliseerde gevalle gevind is nie.

Die vagina het blind geëindig; by die een pasiënt direk agter die himen waar 'n kunsmatige vagina gemaak moes word, en by die ander met 'n 2-duim diepte. In albei gevalle was die himen aanwesig.

5. *Inwendige geslagsorgane.* Geen inwendige genitalia was teenwoordig nie. Die cervix was afwesig en die vagina het in elke geval blind geëindig. Laparotomie in die 2 pasiënte het volledige afwesigheid van die uterus en buise getoon. 'n Horisontale peritoneale vou tussen die rectum en die blaas, soos beskryf deur Goldberg en Maxwell, is nie by die gevalle gedemonstreer nie. Een geval het by laparotomie 'n intra-abdominale gonade getoon met hieraan 'n normaal voorkomende infundibulo-pelviese band en 'n ronde band wat tot die inwendige lieskanaal gestrek het. Reste van Mülleriaanse oorsprong is nie gevind soos by sommige gevalle beskryf is nie.

Primêre amenoree is altyd 'n kenmerk van testikulêre vervrouliking.

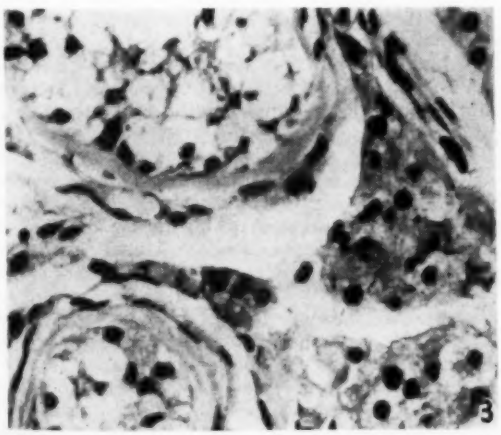
6. *Die gonades.* Die een pasiënt het albei gonades in die lieskanaal gehad, terwyl die ander pasiënt een gonade in die lieskanaal gehad het en die ander gonade intra-abdominaal in die normale posisie van die ovarium. Slegs die pasiënt met albei gonades in die lieskanale het meegaande breuke



Afb. 2. Hipertrofiese klitoris. Normale vulva en blindeindigende kort vagina.

getoon soos dikwels die geval hier is. Die inguinale gonades het pyn veroorsaak en moes verwyder word.

Die dikwels beskrewe tubulêre adenome is nie by hierdie gevalle gevind nie. Die histologiese voorkoms van die gonades was tipies; die gonades het hoofsaaklik bestaan uit onrype seminifereuse buise met interstisiële sel-hiperplasie. Die kapsel van kollagene weefsel was dik en die hele histologiese voorkoms was dié van onryp onafgedaalde testes (Afb. 3). Interessant was dat die intra-abdominale testis in die een geval veel meer interstisiële sel-hiperplasie getoon het as dié



Afb. 3. Histologiese voorkoms van die gonade met Leydig-sel-hiperplasie.

in die lieskanaal; dit mag dui op die rol van intra-abdominale hitte aangesien hierdie interstisiële sel-hiperplasie ook uitgesproke is by die onafgedaalde normale testis.

7. *Hormonale status.* (*a*) Estrogeen. Estrogeenbepalings is nie in die gevalle gedoen nie. Sitologiese ondersoek van vaginale uitstryk-preparate het in die jong pasiënte 'n estrogeen-effek getoon ook na kastrasie. Die estrogeniese aktiwiteit van die testes is egter aangetoon deur menopousale simptome in die pasiënte na kastrasie. Hierdie simptome het goed gereageer op estrogeen-toediening. Die ontwikkeling van die vroulike mammae in die gevalle is te danke aan die effek van estrogeen.

(*b*) Androgeen. Die 17-ketosteroïde was binne die normale perke in albei gevalle na kastrasie. Voor kastrasie is slegs een bepaling gedoen, maar Wilkens en sy medewerkers[11] kon by hulle gevalle die afskeiding van sowel androgeen as estrogeen van die testes aantoon.

Voor die operasie was die 17-ketosteroïde-uitskeiding per 24 uur in die een geval 15·1 mg. en na die operasie was dit 6·8 mg., in die ander geval was die 17-ketosteroïde na die operasie 5·2 mg. in 24 uur se urine.

(*c*) Gonadotropien. Die F.S.H.-waardes was na kastrasie verhoog bo 48 muiseenhede by albei pasiënte. Voor kastrasie is nie F.S.H.-bepalings uitgevoer in die gevalle nie, maar hoë waardes word ook hier gemeld in die literatuur, en Morris[1] voer aan dat die estrogene deur die testes gevorm, kwalitatief mag verskil van normaal gevormde estrogeen, en nie die hipofese rem nie. Prader het bevind dat die steroïde hormoonwaardes van die urine in sy gevalle, sowel as hul gonadale histologie, behalwe vir hoë estrogeenwaardes, nie te onderskei was van normale kriptorgidisme nie.

8. *Kastrasie.* Voor puberteit onderdruk kastrasie die ontwikkeling van die vroulike mammae, maar vermanliking kom nie voor nie en die persone bly vroulik. By die volwassene gee kastrasie aanleiding tot estrogeen-onttrekkingsverskynsels soos atrofie van die vaginale epiteel, warm gloede, verkleining van die mammae, verlaging van die urinêre estrogeen-gehalte en verhoging van die F.S.H.-waardes.

Van hierdie veranderinge was slegs die warm gloede uitgesproke by die 2 pasiënte. Vetsug het by een pasiënt voorgekom na kastrasie.

9. *Chromatienpatroon.* Die chromatienpatroon was manlik in albei gevalle.

Diagnose

Gevalle met estrogeen produserende testes verskil oënskynlik minder van geneties-normale vroue as van die ander intersekse. Baie gevalle van hierdie sindroom word waarskynlik verkeerdelik gediagnoseer as uterien-anomalië of ovaria in liesbreuke.

Waar hierdie anomalië vermoed word van die geskiedenis en 'n deeglike bekkenondersoek verrig word met bepaling van die chromatienstruktuur, hormoonbepalings en biopsie van die gonades, behoort die diagnose nie misgekyk te word nie.

In sommige gevalle mag die vervrouliking minder volledig wees met vergroting van die klitoris of gedeeltelike vereniging van die labia. Soms mag 'n urogenitale sinus bestaan wat verbind is met 'n vaginale sak, of die Mülleriaanse sisteem mag gedeeltelik ontwikkel wees en nie totaal ontbreek nie.

Met deeglike ondersoek kan hierdie gevalle egter ook korrek gediagnoseer word.

Behandeling

Herstel van testikulêre vervrouliking bestaan nie. Die pasiënte kom gewoonlik om advies weens steriliteit, primêre amenoree of liesgeswelle en breuke. Meestal word hulle dus eers na puberteit ondersoek.

Word die diagnose gemaak voor die puberteit vanweë 'n liesbreuk en die tipiese makroskopiese voorkoms van die testes, moet die gonades nie verwyder word nie. Die breuk moet herstel word met terugplasing van die gonade in die buikholte na inspeksie van die bekkenorgane en veral die ander gonade. Biopsies moet geneem word van die gonades om die diagnose te staaf.

'n Hoë voorkoms van neoplasie in hierdie gonades na die puberteit (20% van 82 gevalle waarvan 7 kwaadaardige gewasse was[1]) noodsaak hul verwydering na die adolessensie. Neoplastiese veranderinge kom nie voor die puberteit voor nie en die teenwoordigheid van die gonades is essensieel vir die ontwikkeling van die vroulike sekondêre geslagskenmerke. Na verwydering van die estrogeen produserende testes is vervangingsterapie met estrogene-preparate per mond aangewese.

Waar die diagnose by 'n volwassene gemaak word, word die testes om dieselfde redes verwyder en vervangingsterapie word dan gegee. Vaginoplastie mag nodig wees in gevalle waar die vagina afwesig is of te kort is vir normale geslagsomgang.

Die voorkoming van psigiese trauma by hierdie pasiënte is van die uiterste belang en 'n volledige uitleg aan die pasiënt van die patogenese van haar toestand dien onder geen omstandighede enige ander doel as om die pasiënt in vertwyfeling te bring nie. Die pasiënte moet verseker word van hulle volwaardigheid as vroue met normale huweliksmoontlikhede, behalwe dat hulle nie kinders sal hê nie.

Onder geskikte omstandighede kan die pasiënte aangemoedig word om kinders aan te neem en sekerlik moet hierdie goed aangepaste en volkome vroulike pasiënte nie huweliksregte ontsê word nie.

Graag spreek ek my waardering uit teenoor prof. F. G. Geldenhuys dat hy my toegelaat het om die gevalle te hanteer asook vir sy simpatieke hulp en advies. My dank aan dr. P. N. Swanepoel, Superintendent van die Algemene Hospitaal, Pretoria, vir verlof om die gevalle te publiseer.

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THE EFFECTS OF SYMPHYSIOTOMY ON THE BANTU FEMALE SACRO-ILIAC JOINT*

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An unbiased attempt to evaluate the effects of lateral separation of the pubic rami on the posterior pelvic joints requires avoidance of generalizations. It is therefore proposed to deal only with the obstetrical operation of symphysiotomy on the full-term pregnant Bantu female.

It is dangerous to be dogmatic about the sacro-iliac joint. Any assessment of permanent injury or damage to this articulation needs intimate knowledge of the many variations from the normal, if indeed there is a norm, exhibited by this joint in its healthy state. What are the factors to be considered?

1. *Classification*

The joint does not admit of satisfactory anatomical classification, as witness the variety of descriptions.[9, 10, 11] Smout and Jacoby's ingenious but clumsy classification, namely as a diarthrodial heteromorphic atypical hinge joint, is even inadequate. Phylogenetically, the obstetrical function is subordinated to the weight-bearing function in man.[7] Nevertheless, the remnant obstetrical function influences the joint to such a degree that sexual differentiation, especially in regard to mobility, is pronounced.

It has been clearly shown that the elderly male possesses a physiologically ankylosed joint,[3, 4, 8, 14] whereas its female multiparous counterpart has joint mobility equalling or in excess of that of young males or nulliparae. The 75-year-old male therefore has a joint which is a synarthrosis, and the 75-year-old multiparous female is in possession of a diarthrosis. It follows, therefore, that evidence of commencing ankylosis on the articular surfaces of middle-aged males, especially near the dorsal border of an auricle, is no indication of joint pathology. This naturally also applies to the nulliparous female joint.

Any interpretation of bony changes on the articular aspects of Bantu joints must apparently be accompanied by extreme circumspection. In the South African Bantu there exists a noticeable and apparently inconsistent variety of morphological patterns of joint architecture.[12] In Weisl's study[11] of the articular surfaces of the sacro-iliac joint no mention is made of morphological inconsistency; this investigation was carried out on Europeans and the question of the existence of racial differentiation becomes actual.

2. *Bony Details*

Roentgenological studies of pelvic trabeculation on the basis of the Meyer-Wolff law of transformation of bone lends impressive support to the supposition that the sacro-iliac joints are the weak links on the bony supporting arch of the pelvis. From this the possibly fallacious conclusion can be reached, that the small bony shelf on the ventral border of the iliac auricular surface supports the arch of the pelvic bridge.

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The Meyer-Wolff law, interpreted in terms of trabecular patterns only, is open to serious criticism in view of the impressive experimental work by Honor B. Fell.[6, 5] The weak bony ridge or shelf on the lower border of the iliac articular surface appears to be of recent origin, and is most probably the result of bone apposition according to Roux's principle.[13] It is possibly an overflow of thwarted growth due to forceful obstruction from the ventral edge of the sacral auricular surface at the time of increased pubertal growth activity.[7, 12] Brailsford's observation[1, 2] on increased pronunciation of the pre-auricular sulci in multiparae can be explained by further stimulation of bone apposition on this shelf of bone as a result of tension within physiological limits. The direction of this tension completely invalidates any theory of supportive function of this small ridge.

3. *Ligaments and Accessory Ligaments*

Microscopical studies show that under the influence of 'relaxin' during pregnancy fundamental changes occur in collagenous structures. It is not my intention in a brief paper to pursue the complexities of this very definite but little-known reaction. Suffice it to state that, presumably as a result of the action of a fraction of the corpus luteum hormones of pregnancy, the collagenous component of the Bantu female symphysis pubis is subject to the following changes (Figs. 1 and 2).

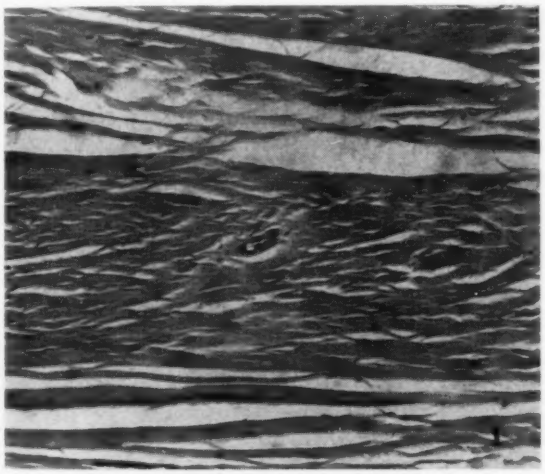

Fig. 1. Photomicrograph of anterior symphysial ligament of Bantu female (non-pregnant) aged 34 years. H and E×120.

(*a*) An approximately 100% increase in thickness.

(*b*) A very noticeably increased vascularization.

(*c*) A marked increase and hypertrophy of individual collagen fibres. This is associated with an appearance of elasticity as compared with the elongated straight and thin fibres of the ligaments not influenced by the hormone. Changes from a small deep-staining pyknotic nucleus to a larger swollen pale-staining nucleus seem to indicate a basic cellular reaction.

There is very good reason to believe that this reaction is not localized to the anterior pelvic joint, but that the whole body is affected by the hormone. If so, then the ligaments and accessory ligaments of the sacro-iliac joint are also affected. Experimental evidence appears to confirm this.[12, 15]

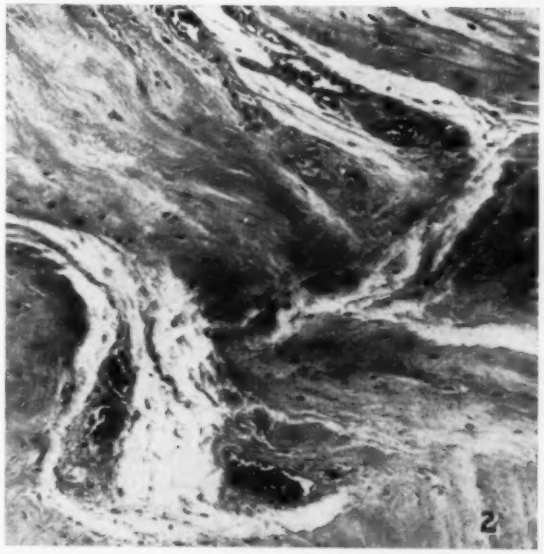

Fig. 2. Photomicrograph of anterior symphysial ligament of pregnant Bantu female aged 38 years. H and E×120.

If, then, the ligamentary changes in the posterior pelvic joints are similar to those in the anterior pelvic joint a case could be argued for greatly diminished control of joint movement by the thin anterior and thick posterior joint ligaments, as well as the more remote and accessory sacro-tuberous and sacrospinous ligaments. The latter two appear to control only very exaggerated movements of the joint. The greatly increased elasticity would also protect against early ligamental damage.

Macroscopically, therefore, during or immediately after pregnancy, it is extremely difficult to estimate whether damage to these ligaments is apparent or real.

4. *Mechanics*

The study of sacro-iliac joint movements is a profound one in its own right. Because of inaccessibility of the joint, statements on mobility in the living subject have for many years depended on deductive evidence. With the advent of precise radiographic techniques impressive studies have been made which show a surprisingly wide range of sacral movements of a rotatory nature.[15]

It is surprising that for nearly a century the importance of the active lateral movement of the innominates on the sacrum has been completely disregarded. Investigations on the mechanics of the joint have mainly been directed towards sacral rotation, despite the fact that the latter can hardly occur without prior ventral widening of the joint. It is perhaps more correct to say that the two movements are nearly synchronous, although the lateral movement is the primary one.

Recent experimental evidence appeared to indicate that this lateral movement is an active one brought about by muscular action.[12] Further radiographic evidence of this movement has since been obtained. It is not proposed at this stage to deal in detail with the methods employed, nor with all the results that were obtained. In the main, previous methods[12] were employed except that additional studies

were made in positions of trunk and thigh flexion, and only symphysial widening was accepted as an absolute indication of lateral movement of the innominates. The following relevant results were obtained:

(*a*) Lateral movement of the innominates at the sacro-iliac joint as a result of muscular action occurred in every one of 35 subjects examined.

(*b*) Where the movement was a requirement of muscular action as a result of trunk extension (33 cases) a positive result was obtained in all 33 cases. Where the movement was a requirement of active trunk and thigh flexion (17 cases) again a positive result was obtained in all 17 cases.

These 100% positive results (*a* and *b*) are deemed statistically significant.

(*c*) The increase in width of the symphysis in 15 Bantu subjects (reflecting in direct proportion the extent of the gaping movement at the sacro-iliac joint) was on an average 39% greater during the act of trunk and thigh flexion than during trunk extension. Actual figures relative to the average increase in symphysial width were 1·52 mm. in extension and 2·11 mm. in trunk and thigh flexion. This ratio is more or less maintained by both multiparae and primiparae. Trunk and thigh flexion is of course associated with expulsive efforts during the second stage of labour.

(*d*) Approximately one-third of the pre-labour X-rays of the 15 Bantu subjects show 'vacuum' formation in the symphysis pubis. These 'vacuums' all disappeared on the subsequent postpartum photographs. As vacuum formation is authoritatively regarded as a sign of traction or tension resulting in separation of articular surfaces,[8] it would seem that there might be noticeable tension in the symphyseal joint during pregnancy. This tension can only be effected by active sacro-iliac widening.

Collectively the above considerations appear to indicate an obstetrical as well as a weight-bearing function in the Bantu female sacro-iliac joint. This function presents primarily as a lateral movement of the two innominates on the sacrum.

Symphysiotomy aims at moving the pubic rami in a lateral direction, i.e. it artificially creates a lateral movement in the sacro-iliac joints. Is this movement, then, within reasonable limits unphysiological? It seems that the answer is no.

OPSOMMING EN GEVOLGTREKKING

1. Anatomiese oorwegings, wat ontogenetiese, mikroskopiese en meganiese studies insluit, dui op 'n obstetriese werking by die iliosakrale gewrig van die vroulike Bantoe.

2. Hierdie funksie word grotendeels geopenbaar deur 'n dwarsbeweging van die innominata op die kruisbeen.

3. Die verloskundige ingreep van simfisiotomie bewerk hierdie beweging kunsmatig. Binne redelike perke is hierdie beweging nie onnatuurlik en ook nie skadelik nie.

I wish to express appreciation to Prof. O. S. Heyns for kindly granting permission for and assistance with the radiological investigations at the Bridgeman Memorial Hospital, to Dr. J. M. Samson, Superintendent of the Bridgeman Memorial Hospital, for time and assistance with the X-ray investigations, and to Prof. P. V. Tobias of the Department of Anatomy, University of the Witwatersrand, for the use of skeletal material.

Ook my dank aan Dr. H. Rompel, Geneeskundige Superintendent van die Suid-Randse Hospitaal, vir raad en daad met die fotografiese opnames.

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WORLD LIST OF INTERNATIONAL MEETINGS

Eighth European Meeting on Electroencephalographic Information, Marseilles, October 1959. Dr. R. Naquet, Hôpital de Timone, Marseilles, France.

Society of French-speaking Comparative Pathologists, 9th Conference, Bordeaux, France, October 1959. Dr. L. Grollet, 7 rue Gustave-Nadaud, Paris 16e, France.

Sixth International Congress of Therapeutics, Strasbourg, 1-3 October 1959. Prof. F. Schmidt, Secretary, c/o Institut de Pharmacologie, Hôpital Civil, 1 place de l'Hôpital, Strasbourg (Bas-Rhin), France.

Fourth International Medical Congress, Verona, 1-4 October 1959. Secrétariat, Journées Médicales Internationales, c/o Instituti Ospitalieri, Borgo Trento, Verona, Italy.

Second International Colloqium on Congenital Malformations of the Skull, Paris, 2-3 October 1959. Dr. Michel Feld, 6 rue de Villersexel, Paris 7e, France.

First International Congress of Histotherapy and Cellular Therapy, Wiesbaden, Germany, 2-4 October 1959. Dr. R. Henry, 54 rue Beauborg, Paris 3e, France.

World Health Organization, Expert Committee on specifications for pharmaceutical preparations, Sub-committee on non-proprietary names, Geneva, 5-7 October 1959. Palais des Nations, Geneva, Switzerland.

French-Language Association of Scientific Psychology, 6th Session, Bordeaux, 5-8 October 1959. Prof. Chateau, Institut de Psychologie, 20 cours Pasteur, Bordeaux, France.

World Health Organization, Seminar on the rehabilitation of physically handicapped children, Barcelona, 5-10 October 1959. Regional Office for Europe, WHO, 8 Scherfigsvej, Copenhagen Ø, Denmark.

Fifth International Convention on Nutrition and Vital Substances, Constance, Germany, and Zurich, 7-11 October 1959. Dr. S. Klein, 29 square Larousse, Brussels, Belgium.

Pan American Seminar on Pharmaceutical Education, San Juan, P.R., 11-17 October 1959. Dr. Melvin W. Green, American Council on Pharmaceutical Education, 77 W. Washington Street, Chicago 2, Illinois.

Seventeenth Congress of French-Speaking Paediatricians, Montpellier, 12-14 October 1959. Prof. Roger Jean, Secretary General, 8 rue Guillaume-de-Nogaret, Montpellier (Herault), France.

World Health Organization, European Technical Conference on the contribution of the medical officer to the psychosocial environment in industry, London, 12-16 October 1959. Regional Office for Europe, WHO, 8 Scherfigsvej, Copenhagen ø, Denmark.

World Health Organization, Conference on post-basic nursing education programme for international students, Geneva, 12-23 October 1959. Palais des Nations, Geneva, Switzerland.

International Union against the Venereal Diseases and the Tre-

ponematoses, 32nd General Assembly, London 13-17 October 1959. Prof. G. A. Canaperia, Secretary General, Via Salaria 237, Rome, Italy.

International Venereal Diseases Congress, Meeting of committees, London, 13-17 October 1959. (By invitation.) Dr. G. E. W. Wolstenholme, 41 Portland Place, London, W. 1.

Pan American Medical Association, 10th Inter-American Congress, Mexico, D.F., 19-31 October 1959. Dr. Joseph Eller, Director General, Pan American Medical Association, 745 Fifth Ave., New York 22, N.Y.

Conference on the Control of Infectious Diseases by Vaccination Programmes, Morocco, 23-31 October 1959. (By invitation.) Regional Committee for Europe, WHO, 8 Scherfigsvej, Copenhagen Ø, Denmark.

Pan American Sanitary Bureau/World Health Organization, 5th Regional Nursing Congress, Buenos Aires, 25-31 October 1959. Division of Education and Training, Pan American Sanitary Bureau, Washington 6, D.C.

World Health Organization, Committee on international quarantine, Geneva, 26-31 October 1959. Palais des Nations, Geneva, Switzerland.

Congress of Neurological Surgeons, Miami Beach, 29-31 October 1959. Dr. R. DeSaussure, 899 Madison Ave., Memphis, Tennessee.

International Atomic Energy Agency, Conference on disposal of radioactive waste, Monaco, November 1959. Kaertnerring, Vienna I.

International Centre for Pesticides, General Assembly, Milan, November 1959. 24 Beethovenstrasse, Zurich, Switzerland.

International Medical Days, Nice, November 1959. Dr. Louis Barraya, 51 rue du Maréchal-Joffre, Nice, France.

South Pacific Commission, Study group on filariasis, Noumea, New Caledonia, 2nd half November 1959. Postbox No. 9, Noumea, New Caledonia.

Collegium Internationale Allergologicum, 4th Symposium, Rome, 2-6 November 1959. Dr. A. Cerletti, Pharmacological Laboratories, Sandoz Ltd., Basle, Switzerland.

World Health Organization, Committee on addiction-producing drugs, Geneva, 2-7 November 1959. Palais des Nations, Geneva, Switzerland.

Seventh Annual Symposium on Antibiotics, Washington, D.C., 5-7 November 1959. Dr. Henry Welch, Chairman, c/o Division of Antibiotics, Food and Drug Administration, Washington 25, D.C.

Darwin Centennial Celebration, Chicago, 22-29 November 1959. Dr. Sol Tax, Department of Anthropolgy, University of Chicago, Chicago 37, Illinois.

French-speaking Neurosurgeons Society, Annual Meeting, Paris, 30 November-2 December 1959. Dr. Marcel David, 4 rue Galliera, Paris, France.

World Health Organization (Food and Agriculture Organization of the United Nations), Technical meeting on food additives, Geneva or Rome, December 1959, F.A.O., Viale delle Terme di Caracalla, Rome, Italy.

First Asian Congress International Federation of Catholic Physicians, Manila, December 1959. Dr. R. Verly, 5 rue Guimard, Brussels, Belgium.

European Symposium on Training and Education in Nutrition, Frankfurt am Main, 3-11 December 1959. Food and Agriculture Organization of the United Nations, Viale delle Terme di Caracalla, Rome, Italy.

World Health Organization, Meeting of the Expert Committee on rabies, Geneva, 14-19 December 1959. Palais des Nations, Geneva, Switzerland.

IN MEMORIAM

HYMAN SOLOMON ROSEMAN, M.D., (Dubl.), M.R.C.P. (Lond.)

Dr. Lewis S. Robertson, of Johannesburg, writes: Lt.-Col. H. S. Roseman, of Johannesburg, died on 3 August 1959. I have known him for the past 35 years and became intimately acquainted with him during the war years 1939–1945.

Dr. Roseman

It was my good fortune to have worked with him as my Second-in-Command at the South African Military Hospital for 4 years during the Second World War.

I greatly appreciated his conscientiousness, efficiency, devotion to duty, and his great loyalty. It was my privilege to have had him available for consultation in the many problems which arose at the Hospital during the war period.

In addition to his administrative duties he was Officer-in-Charge of the Medical Division of the Military Hospital, and under his guidance and control a most efficient medical service of the highest order was established at the Hospital.

Lt.-Col. Roseman had a pleasing personality, was popular with his colleagues, and had a keen sense of humour. But it was his patients who appreciated him more than anyone else, and I was aware of the high esteem in which he was held by them.

Lt.-Col. Roseman and I remained staunch friends after the war years and I shall greatly miss a truly genuine colleague and friend.

I have known Mrs. Roseman since the time of her wedding and have been greatly interested in the two daughters, of whom their late father was so justly proud.

The large attendance at the funeral service on 5 August 1959 was evidence of the high esteem in which my late colleague and friend was held in Pretoria.

Dr. J. H. Struthers, Chairman of the Federal Council of the Medical Association, writes: Dr. Roseman took a very active interest in the affairs of the Medical Association of South Africa, and proved himself a very reliable colleague. He was elected a member of the Northern Transvaal Branch Council in 1940, and remained a member until he retired from active practice. He became President of the Branch in 1948, and made a considerable contribution to the affairs of the Association—particularly in the struggle with the Province over free hospitalization.

He was an original thinker, always likely to produce constructive ideas—he had a keen sense of humour and was a man of great loyalties. Even in his latter years he always evinced great interest in the affairs of the Association and loved to discuss them. He will be sadly missed by his colleagues in Pretoria.

Dr. Ben Epstein, of Pretoria, writes: Dr. H. S. Roseman was born in 1898. He came to South Africa as a child of 4 years and, except for the periods when he was studying overseas, he lived in Pretoria all his life. In 1919 he enrolled as a medical student at the University of Cape Town, where he completed his first year. He received the rest of his training in Dublin where he qualified in 1923. He was, for some years, in general practice in Pretoria, and built up a large practice. In 1930 he gave up general practice, and proceeded overseas for postgraduate study in Great Britain and the Continent. In 1932 he obtained the M.D. (Dubl.) degree, and in the following year he was admitted as a Member of the Royal College of Physicians (London).

His return to Pretoria in 1934 was a milestone in the city's medical history. He was the first doctor to practice as a specialist physician, and his presence made an immediate impact on the standards of medicine in Pretoria. It is a long way from these years to the present day, when we have large numbers of specialists in all branches of medicine.

Hymie Roseman set himself a high standard in his work and in his life as a consultant physician. He was a natural teacher, and it was a pleasure to call him into consultation, and to see him take a history and examine a patient with the meticulous care for which he was so well known. In discussion of the case

afterwards he presented the facts with amazing lucidity. In his talks with patients and their families he invariably put the general practitioner in the foreground, and gave him all the support possible.

He held a number of professional appointments. He was an Assistant Honorary Physician to the Pretoria Hospital. He was part-time Consultant Physician to the No. 1 South Africa Military Hospital at Voortrekkerhoogte before the last war, and at the outbreak of hostilities he joined up for full-time military service. He rose to the rank of Lieutenant-Colonel and, at the end of the war, he received a Royal commendation for his services. In 1947 he was appointed physician to the Royal family during their visit to South Africa.

Hymie Roseman has been an active member of various medical societies in Pretoria. In 1948 the Northern Transvaal Branch of the Medical Association honoured him by electing him its President—at a turbulent time in the affairs of this Branch. He took an active interest in the settlement of ex-service men, and became the first chairman of the Jewish Ex-Servicemen's League in Pretoria.

Failing health and the pressure of business interests forced him to abandon medical practice in 1955, and restrict himself to business. This was a difficult decision for him to take, and he never really abandoned his interest in medicine and in the Medical Association. He was always interested in medical 'shop', both scientific and personal.

Hymie Roseman was a man of exceptional intelligence and common sense. He had an excellent sense of humour and was an extremely good raconteur; at social gatherings he was usually the centre of an interested group. He was a staunch supporter of democratic principles, and his sharp tongue was at its best when he was fighting for what he thought to be right and just. For his many friends he was always prepared, when required, to take endless trouble.

He was a most devoted husband and father. His affection for his family and his gentleness with them, were among his outstanding attributes. His lovely home, with its beautiful view of Pretoria and the distant hills, is now a place of sadness. His widow Hilda, and his daughters Dawn and Doreen, have as consolation the memory of a man who was the essence of kindness, who guided them in their difficulties, and who made their lives his own.

JOHANNES NICOLAAS WILLEM LOUBSER, B.A., M.D., CH.D. (BERLIN)

Dr. James Gilliland, Fre-sekretaris, Noord-Oostelike Afdeling van die Tak Oranie-Vrystaat en Basoetoland (M.V.S.A.), Bethlehem, skryf: Dr. J. N. W. Loubser is op 5 Augustus 1959 in die ouderdom van 79 jaar op Bethlehem oorlede. Hy is op 8 Maart 1881 te 'Koeberg' in die distrik van Malmesbury, Kaap, gebore. Sy skoolopleiding het hy op Stellenbosch geniet. In 1888 is hy na die Rhenish Instituut, daarna na die Stellenbosch Gimnasium, en toe na die Victoria Kollege, waar hy in Februarie 1902 die B.A.-graad met onderskeiding behaal het.

Dr. Loubser

In Mei 1902 het hy as mediese student ingeskrywe aan die Universiteit van Strassburg (Duitsland), en daar het hy in 1905 sy eerste mediese eksamen afgelê. Hy het toe een semester in München gestudeer en daarna na die Universiteit van Berlyn gegaan, waar hy in Junie 1907 die Staatseksamen afgelê het. In Desember van dieselfde jaar het hy die grade M.D., Ch.D. *cum laude*, behaal met 'n proefskrif getitel: 'Implantationsgeschülste der Bauchdecken nach Ovariotmien'.

Vanaf Junie 1907 tot Augustus 1908 het hy in verskillende hospitale diens gedoen, o.a. in die Universiteitskliniek, Halle, vir neus-, keel- en oorsiektes; in die Universiteitskliniek, München, vir chirurgie; en in die Städtisches Krankenhaus, Augsburg.

In September 1908 het hy na Suid-Afrika teruggekeer, en tot die einde van die jaar te Worcester, Kaap, gepraktiseer.

In Mei 1909 het hy hom op Bethlehem gevestig nadat hy in die huwelik getree het met mej. Estelle de Villiers.

Gedurende 1925–26 het hy weer oorsee gegaan vir studiedoeleindes. Hy het 6 maande onder professor Merckel, in München, geregtelike medisyne, en 6 maande aan die Tropiese Instituut in Hamburg, gestudeer.

Vanaf 1927 tot 1942 was hy Distriksgeneesheer van Bethlehem.

In 1942 het hy hom van die privaat-praktyk onttrek en het toe 'n aanstelling aanvaar as voltydse Mediese Gesondheidsbeampte van Bethlehem. Hierdie amp het hy beklee tot 1947.

In die samelewing van Bethlehem het dr. Loubser altyd 'n leidende aandeel geneem. Hy was een van die stigters van die hospitaal alhier, en het tot 3 maande voor sy dood nog op die hospitaalraad gedien.

Die ontwikkeling van die mooi Pretoriuskloof, wat een van Bethlehem se aantreklikhede is, is grootliks aan sy ywer en daad te danke. Onder sy pasiënte was dr. Loubser altyd besonder gewild. Hy het nooit enige moeite ontsien vir hulle nie, en sy kalme, innemende geaardheid was altyd 'n steunpilaar vir menige gesin in Bethlehem. Hy was by uitstek die egte familiedokter.

In die Verenigingslewe van die mediese professie het dr. Loubser altyd 'n leidende aandeel geneem. Hy is gekies as sekretaris/tesourier op die stigtersvergadering van die Noord-Oostelike Afdeling (Tak O.V.S. en B.) van die Mediese Vereniging, op 23 Augustus 1928. Hierdie amp het hy beklee oor 'n tydperk van 27 agtereenvolgende jare, totdat hy in 1955, op eie versoek, bedank het.

Vanaf 1929 tot 1941 was hy lid van die Federale Raad van die Mediese Vereniging as verteenwoordiger van die Tak O.V.S. en B.

Hy is verkies tot President van die Tak O.V.S. en B. gedurende 1933 en weer gedurende 1951.

Dr. Loubser was die gekose verteenwoordiger van die O.V.S. op die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad oor 'n tydperk van 10 jaar (1948–1958). Hy het ook op die Uitvoerende Komitee van die Mediese Raad gedien. Gedurende 1959 is die Brons-Medalje van die Mediese Vereniging aan hom toegeken.

Gedurende die 50 jaar wat dr. Loubser op Bethlehem praktiseer het, het hy altyd in volle mate die respek en hoë agting van sy pasiënte, sy kollegas en die algemene publiek geniet. Hy was deel van die geskiedenis van sy omgewing en het altyd die hoogste ideale van sy professie uitgeleef. Sy vrou het hom ontval vroeër in die jaar, na 'n huwelikslewe van 50 jaar. Hy laat een dogter na, mej. Ninon Loubser, aan wie ons ons innige simpatie toewens.

Dr. D. Serfontein, van Heilbron, skryf: Met die dood van dr. J. N. W. Loubser verloor nie alleen die Tak O.V.S. en B. van die Mediese Vereniging nie, maar ook die hele mediese professie in ons land 'n uitstekende medikus sowel as 'n mens met besonder hoogstaande eienskappe. As vriend en kameraad, en as jarelange vennoot en medewerker in die belange van die Mediese Vereniging van Suid-Afrika wil ek graag in 'n paar woorde hulde bring aan ons ontslape kollega.

Jan Loubser het geweet om 'n wetenskaplike *weltanschauung* te verenig met 'n humanistiese (en humanitêre) uitkyk op sake en persone. In die donker dae van die Rebellie het hy byvoorbeeld 'n bloeiende praktyk te Bethlehem verlaat om hom as enigste medikus, en te perd nog, daardie dae, by die rebellemagte aan te sluit. Hy was 'n wydbelese kultuurmens en 'n selfstandige denker, met 'n klinkklare en vinnige brein; en sy raak gesegdes, sy gevatte antwoorde en sy kwinkslae oor mense en dinge sal nog lank as tradisie voortleef by hulle wat hom intiem geken het. Hy was eerlik en snydend reguit, maar het nogtans altyd, sowel as medikus en as mens, 'n warm hart gehad teenoor die minder bevoorregte, die swaar beproefde, of dit nou rasgenoot was of nie. Sy energie en werklus was spreekwoordelik; sy humorsin, ten spyte van swaar terugslae in sy gesondheid die laaste jare, altyd ononderdrukbaar; en sy hulpvaardigheid met raad en daad teenoor almal en veral teenoor jong beginnende medici, was alombekend. Altyd lojaal teenoor sy vriende, sy pasiënte, sy professie, sy stad, sy land en volk voorwaar, 'n pragmens en medikus, van wie ons kan sê dat sy lewe mooi afgerond was in diens van almal.

Ons sal in ons omgang en in ons Verenigingslewe die leemte wat sy dood gebring het altyd gevoelig ondervind. Mag hy vir ons nie alleen 'n herinnering en 'n voorbeeld bly nie, maar ook 'n inspirasie.

WILLIAM CHARLES FONE

Mr. William Charles Fone, until recently editorial assistant on the staff of this *Journal*, died on 19 August 1959, at the age of 72, at his home in Pinelands, Cape.

Mr. Fone, who was born in the West of England, worked as a journalist for over 55 years. While employed on the *Daily Mail*, London, he joined the *Star*, Johannesburg, as a sub-editor, which brought him to South Africa in 1922. In 1924 he became chief sub-editor of the *Cape Argus*, of which he was appointed an assistant editor in 1946, retiring in 1953. From that time until the end of 1958 he worked on the *South African Medical Journal*.

During his 5 years' service with the *Journal* Mr. Fone's friendliness, imperturbability and humour greatly endeared him to his colleagues, and his ripe experience and wise judgment were much valued.

Mr. Fone was a keen sportsman and a lifelong golfer. He leaves his wife, two sons (Mr. R. Fone, Town Engineer, Vereeniging, and Mr. A. Fone, at present in Britain) and a daughter (Mrs. R. Bowie).

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES REMOVED FROM THE LIST

The names of the following medical aid societies have been removed from the list of approved medical aid societies, as from the dates indicated. The members of these societies are, therefore, no longer entitled to the preferential tariff:

Greaterman's Medical Aid Society, P.O. Box 5460, Johannesburg (1 September 1959).
Medical Aid Society for Transvaal Teachers, P.O. Box 28, Boksburg (1 September 1959).
Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg (1 August 1959).
Elwamba Medical Aid Fund, P.O. Box 42, East London.

Medical House
Cape Town
21 August 1959

L. M. Marchand
Associate Secretary

MEDIESE HULPVERENIGINGS VAN DIE LYS GESKRAP

Die name van die volgende mediese hulpverenigings is van die lys van goedgekeurde mediese hulpverenigings geskrap van die die datums af soos aangedui. Lede van dié verenigings is dus nie langer op die voorkeurtarief geregtig nie:

Mediese Huis
Kaapstad
21 Augustus 1959

L. M. Marchand
Medesekretaris

PASSING EVENTS : IN DIE VERBYGAAN

Correction. The letter on 'Cerebral palsy' by Dr. Ben Epstein, published in the *Journal* of 15 August, was written in his private capacity and not as Chairman of the Board of Management of the Pretoria School for Cerebral Palsy (**33**, 695).

In the heading to the copy of the letter to the Chief Inspector of Schools, Transvaal, also published on 15 August, requesting investigation into the problem of home teaching, Dr. Epstein was inadvertently referred to as the Chairman of the South African Orthopaedic Association. This should, of course, read Chairman of the *South African Paediatric Association* (M.A.S.A.) (**33**, 696).

* * *

A History of Medicine in South Africa. Mr. A. A. Balkema, publisher of *A History of Medicine in South Africa*, has requested that the *Journal* inform members of the Association that as the book is being posted in batches to certain areas at a time, there is likely to be some delay before all members receive their copies. Should any member require his copy urgently, he is requested to write direct to Mr. Balkema, Union House, Queen Victoria Street, Cape Town, and his copy will be dispatched immediately.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

Dr. Sidney Stein, accompanied by his wife, has left Cape Town on a 3½-month tour of the USA and the UK, where he will visit various dermatological centres and attend, among other meetings, a dermatological congress at Lake Arrowhead, USA, and the Pacific Dermatological Meeting in California.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Tuesday 15 September at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Prof. J. G. Thomson will speak on 'Primary carcinoma of the liver in the three ethnic groups in Cape Town'. All who are interested are invited to attend this meeting.

* * *

The Alexander von Humboldt-Stiftung awards scholarships for postgraduate study at universities and research institutes in the Federal Republic of Germany and West Berlin. Applicants must not be older than 30 years and should have an adequate command of the German language. The scholarship provides for an amount of approximately £51 per month for board and lodging; fees are paid by the Stiftung. The scholarships are awarded in general for a period of 10 months for the academic year beginning 1 October and ending on 31 July. Enquiries and requests for application forms must be directed to Mr. O. M. Neubert, Embassy of the Federal Republic of West Germany, P.O. Box 2023, Pretoria (telephone 3–5291). Completed application forms must be sent to the abovementioned address before 15 October 1959.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

CHLOROMYCETIN SUCCINATE

Parke, Davis Laboratories (Pty.) Ltd. have introduced Chloromycetin Succinate, a new '3-way' parental form of chloromycetin, which is a readily available and exceptionally well-tolerated form of broad-spectrum antibiotic agent suitable for administration by any of the 3 parenteral routes—intravenous, intramuscular and subcutaneous. Chloromycetin Succinate can also be given by inhalation in certain respiratory conditions.

Description: Chloromycetin Succinate is the sodium salt of the monosuccinic ester of chloromycetin. It is highly soluble in water and parenteral fluids and so is easily prepared for use. It produces minimal tissue reaction at the injection site. Once injected, the ester is rapidly hydrolysed by tissue enzymes, giving effective therapeutic concentrations of free and active chloromycetin within the body.

Indications: Chloromycetin Succinate is active against the usual wide range of chloromycetin-sensitive infections. The fact that adequate therapeutic blood levels can be maintained with almost complete absence of local tissue reactions make Chloromycetin Succinate particularly well suited to paediatric use.

Chloromycetin Succinate also lends itself well to the treatment of patients with enteric disease in its acute phase, when nausea, vomiting or severe diarrhoea might interfere with the effective absorption of oral medication.

An excellent agent for the treatment of various meningitides, including *H. influenzae* meningitis, Chloromycetin Succinate has been reported highly effective in the treatment of a number of respiratory infections, including acute tracheobronchitis, asthmatic bronchitis, and bronchial and pneumonococcal pneumonia. In the last mentioned indications, it is of particular interest to know that the high solubility and rapid hydrolysis of chloromycetin makes possible its use as an aerosol spray in the relief of chronic or subacute respiratory illness with bacterial infection.

Dosage and administration: The gelsiccated powder in the steri-vial is prepared for injection by the addition of an aqueous diluent such as water for injection or 5% dextrose injection. Full instructions for the preparation of a solution accompany each vial, together with recommended dosage schedules for the various age groups.

Package information: Chloromycetin Succinate is supplied in 10 c.c. steri-vials, each containing the equivalent of 1 g. of chloromycetin.

ALCOS-ANAL

Westdene Products (Pty.) Ltd. announce the introduction of Alcos-Anal, manufactured by the Camden Chemical Company Ltd. of London, and supply the following information:

Alcos-Anal represents a completely new concept in the therapy of haemorrhoids. Although applied in the same manner as substances providing only symptomatic relief, Alcos-Anal is, in addition, an actual treatment for this condition. The active ingredient is sodium salts of highly unsaturated fatty acids incorporated in a base which increases permeability of the mucous membrane. These fatty acids produce muscular contraction and cause increased growth of the collagen fibres into the vascular walls. This leads to subsequent shrinking of the nodules which diminish in size to a point where in many cases they are no longer perceptible. Provided treatment is continued relapses rarely occur.

It must be emphasized, however, that permanent relief may be obtained only by a prolonged course of treatment. It is necessary to continue using Alcos-Anal for at least 6 weeks if this is to be achieved. Application at first may cause a burning sensation, but this soon subsides and is never severe enough to warrant discontinuing the treatment. Alcos-Anal is supplied in the form of suppositories in boxes of 10 and 100, and in 20 g. tubes of ointment. Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

ANUSOL H.C.

Warner Pharmaceuticals (Pty.) Ltd. announce the introduction of a new addition to their ethical pharmaceutical line—Anusol H.C.— and supply the following information:

Anusol H.C. is the regular and well known Anusol formula plus 5 mg. of hydrocortisone.

Indications. Anusol H.C. is indicated for the treatment of proctitis, pruritus ani and inflamed haemorrhoids.

Dosage. Two suppositories are given each day, 1 in the morning and 1 at night. Anusol H.C. therapy is continued for 3–6 days, depending on the severity of the inflammation. When Anusol H.C. is discontinued the patient may be switched to Anusol alone for as long as necessary.

Anusol H.C., like Anusol, also soothes and decongests and, because it contains no powerful analgesics, there is no danger of masking symptoms of severe pathology.

BOOK REVIEWS : BOEKBESPREKINGS

CARDIAC ARREST

Cardiac Arrest and Resuscitation. By Hugh E. Stephenson, Jr., M.D. Pp. 378. 31 figures. South African price £5. 2s. 0d. St. Louis: The C. V. Misby Company. 1958.

The author of this very excellent monograph is the Professor of Surgery at the Missouri School of Medicine, USA, and he has made a prolonged and extensive study of cardiac arrest and its treatment. As part of this investigation he established the Cardiac Arrest Registry and his conclusions are based on a study of over 1,700 cases of cardiac arrest from this registry, as well as on work done in the experimental laboratory. A bibliography covering 53 closely printed pages is included. The result is a book of the greatest value to all those who operate under general anaesthesia and to anaesthetists. Professor Stephenson also points out that cardiac arrest occurs during dental extractions and during special radiological procedures, and that in cardiac arrest from these causes a survival is the exception; so it would appear that the specialists concerned here should also be familiar with the contents of this book, and that they should at all times be prepared for this emergency.

The author points out that the number of deaths due to cardiac arrest in the USA exceeds those from poliomyelitis, multiple sclerosis, scarlet fever, typhoid and diphtheria together, and this clearly emphasizes how important this condition has become. It behoves every medical practitioner to make himself absolutely familiar with the management of this condition and there is no better way than to read this book. D.J. du P.

CORRESPONDENCE : BRIEWERUBRIEK

FATAL STATUS ASTHMATICUS

To the Editor: I have latterly observed some 4 cases of status asthmaticus in middle-aged people all with a fatal outcome. This is entirely different from experience gained during previous years, when similar cases, from a clinical point of view, all recovered on expectant therapy.

The fatal cases had all, for some time previously, been treated with one or other corticosteroid. When seen all had low blood pressures which resisted all forms of therapy, with very severe respiratory distress.

No autopsies were possible.

It has occurred to me that one common factor in these cases was their previous treatment with corticosteroids.

It would be interesting to know if other clinicians have had similar experiences.

M. M. Posel

509 Lister Building, Johannesburg
21 August 1959

MEDICAL SERVICES PLAN

To the Editor: The following rule was adopted by the Board of Directors of the Medical Services Plan at its meeting held on 22 July 1959. The rule was subsequently adopted by the Branch Council of the Southern Transvaal Branch of the Medical Association of South Africa, and will be put into immediate effect:

'Where a Subscriber to the Plan or his Dependant is obliged to seek medical services in an area where the Plan does not yet operate, the Plan will treat the Medical Practitioner concerned as a Participating Doctor, for the purposes of payment, provided that the Medical Practitioner shall have agreed with the Subscriber in advance to accept payment from the Plan as a Participating Doctor before rendering services.'

P. J. Parvus
General Manager

Medical Services Plan
P.O. Box 10314
Johannesburg
21 August 1959